THE
LARYNGOSCOPE.

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SYMPOSIUM: OTO-LARYNGOLOGIC CASE REPORTS.†

I.—LARYNGEAL CONDITIONS.*

1. Vocal Nodules.
2. Polypi of Vocal Cords — Two Cases.
3. Epithelioma — Vocal Cord.
4. Carcinoma — Laryngectomy.

DR. JOHN J. MADDEN, Brooklyn.

For centuries medical science has been trying to eradicate from the human race malignant disease.

The larynx is very frequently affected by this condition and the laryngologist is playing a most important part in this great undertaking.

The following few cases tend to illustrate what is being done at our institution in that endeavor; and it is hoped that in future consideration of cases of this nature, the one presenting symptom, hoarseness, will be given its just place as the keynote of warning.

Case 1. White, female, age 26 years, born in United States; admitted to St. Vincent's Hospital Oct. 3, 1934. Complained of hoarseness and decreased voice power for one year. Worse in summer, all right in fall and winter.

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†Program presented by the Staff of St. Vincent's Hospital, New York, Department of Oto-Laryngology.

*Read before the New York Academy of Medicine, Section on Otolaryngology, Jan. 15, 1936.

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Past History: Tonsils and adenoids removed in childhood.

Physical Examination: Pharynx negative. Heart and lungs normal. Blood pressure 116/74. Larynx: Two small white nodules on edge of right cord. Preoperative diagnosis: Singer's nodes.

Operation: Under ether anesthesia and use of Imperatori subglottic forceps, small nodules were removed from edge of vocal cord by grasping the growth in the instrument, gently pulling it from edge of cord, then cutting clean so as to leave the cord unnotched. Operator: Dr. John M. Lore.

Pathologist's Report: "Specimen too small for diagnosis." Discharged Oct. 4, 1934.

Case 2. Mr. H. Van R., white, male, age 50 years, born in Holland, widower; admitted to St. Vincent's Hospital Jan. 17, 1935, with complaint of hoarseness for five or six months. Cough developed after the hoarseness but without hemoptysis.

Past History: Chronic purulent otitis media, right ear, for many years with partial loss of hearing on same side. Admits venereal disease (chancres) many years ago.

Family History: Negative.

Physical Examination: Eyes, nose and central nervous system negative; ears as above; neck, no adenopathy or rigidity. Teeth, upper and lower plates. Heart, negative. Chest expanded poorly; breath sounds poor in volume. Sounds seemed amphoric over left apex. X-ray of chest negative for parenchymal pulmonary pathology; heart, normal in size and position. Sputum, negative for tubercle bacilli. Blood, Wassermann negative. Urine, negative. Larynx: Indirect method, right cord pathologic; nodular mass on both cords.

Preoperative Diagnosis: Polypoid growth, tuberculous or possible syphilitic lesion.

Operation: Gas, oxygen, ether anesthesia, supplemented by cocaine to mucosa of larynx. Larynx exposed by anterior commissure speculum. Polypoid tissue was seen on anterior surface of both cords. Using the Imperatori subglottic forceps the upper surfaces of both cords were stripped, removing the polypoid tissue. (Stripping the cords.) Operator, Dr. John M. Lore.

Pathologist's Report: Grossly, four pieces (small) reddish-white tissue; epithelial surface with cystic submucosa. Microscopically: Thickened stratified squamous epithelium, nonmalignant; benign polyp.

Discharged Jan. 24, 1935.

Case 3. John M., white, male, age 68 years, born in United States; admitted to St. Vincent's Hospital April 13, 1935, as patient of Dr. Lore, complaining of hoarseness for four or five years.

Past History: In past five years he has had two or three operations for removal of growths from larynx.

Physical Examination: Heart and lungs, negative. Larynx showed a papillomatous mass on right cord and anterior commissure regions. There was no lack of motility.

Preoperative Diagnosis: Hyperkeratotic papilloma (benign).

Operation by Dr. John M. Lore: Using 10 per cent cocaine and anterior commissure speculum, a mass attached to anterior third of right cord extending to anterior commissure and subglottically was punched out. Part of right cord was included. Mass seemed hard and bled freely.

Pathologist's Report: Grossly, six small, irregular, firm, homogeneous gray pieces of tissue, all sectioned. Microscopically: Dense connective tissue background extensively infiltrated with cells of basal layer. Some cells with multiple nuclei and atypical mitosis. Diagnosis: Infiltrating squamous cell epithelioma, scirrhus type.

I have since learned that this man was subsequently operated upon by Dr. Brandon at the Manhattan Eye and Ear Hospital. The operation was a successful total laryngectomy and I understand the patient is well and is present here tonight.

Case 4. Mrs. T., white, female, housewife, age 35 years, of French descent, was admitted to St. Vincent's Hospital Dec. 10, 1934, complaining of "huskiness of voice," first noticed seven years previously.

Past History: Laryngeal operation seven years ago and four subsequent laryngeal operations, with biopsy reports of hyperkeratotic papilloma. No accidents.

Physical Examination: Larynx: Large subglottic mass on left side and two small nodules at edge of left cord. Neck: Mobile small gland on left side. Pupils unequal, right smaller than left, both reacting to light and accommodation. Mouth: No lower teeth, upper plate. Chest: X-ray of chest negative, lungs clear; heart, systolic murmur at apex transmitted to axilla; P_2 accentuated.

Treatment: Direct laryngoscopy and biopsy from subglottic region. Tissue was hard and extended fairly well below and into anterior commissure regions.

Pathologist's Report: Squamous cell epithelioma.

Patient discharged Dec. 11, 1934, unimproved.

This case and the previous one illustrate important facts: 1. Hyperkeratotic papilloma is a forerunner of malignancy. 2. In benign looking tumors of the larynx, biopsy and pathologist's report may determine malignancy. 3. Do not be content with a single biopsy having a negative report.

Case 5. Mr. Azar B., age 54 years, born in Syria, occupation, porter; admitted to St. Vincent's Hospital June 4, 1931, complaining of increasing hoarseness which he first noticed four or five months previously. No pain; occasional difficulty in swallowing, patient indicating left side of neck; no difficulty in breathing.

Past history and family history, negative.

Physical Examination: Larynx: Arytenoids swollen and edematous; left cord fixed; right cord moved and met the other beyond the midline; left cord ulcerated. Chest X-ray: Heart within normal limits; aorta, moderate dilatation throughout; no abnormality of esophagus or delay in passage of bismuth meal. Cardiovascular, gastrointestinal, genitourinary, orthopedic and neurological examinations negative. Skeletal muscle, scalp, eyes, ears, nose and pharynx, negative. Blood count and chemistry, normal. Wassermann, negative.

Preoperative Diagnosis: Carcinoma of larynx.

Treatment: The dental service removed a number of infected teeth and proclaimed his mouth free from infection. Patient was operated on, a total laryngectomy being done on June 26, 1931, under rectal anesthesia. Operator: Dr. Madden, assisted by Dr. John D. Kernan.

Operation: Midline incision of neck from thyrohyoid membrane to second ring of trachea, severing skin, soft tissues and platysma. Thyroid cartilage and trachea exposed; thyroid isthmus severed between hemo-

stats; all vessels clamped and ligated. A second skin incision at upper end of first and at right angles to it was made, extending to anterior borders of sternomastoids. All vessels ligated. After full exposure of thyroid cartilage and trachea above and below and sides and all bleeding controlled, trachea was severed transversely at second ring. Then the part of trachea below the section was well freed and drawn through a transverse buttonhole opening made below lower end of original midline incision and anchored to skin with sutures tied over rubber tubing (catheter). The cricothyroid cartilages were dissected upward, using great care in separating from the esophagus not to cut into the esophagus. Above the thyroid cartilage the thyrohyoid membrane was cut transversely, just below the epiglottis, being careful to get plenty of esophagopharyngeal membrane. The thyroid cartilage with the cricoid and first ring of trachea was now excised. Soft rubber duodenal tube next was guided through the nose and into the esophagus and then the esophagopharyngeal membrane was sutured, inverting the mucous surface edges with plain catgut. Sternomastoid muscles and subcutaneous tissues were next sutured to close the exposed area. Two cigarette drains were placed at upper angles of wound and skin was closed with silkworm gut. Tracheotomy tube was put in place and dressings applied.

Postoperative Care: Rectal temperature between 100-103° for nine days, then remained normal. Respirations between 18 and 24; pulse between 70 and 110. Daily dressings for two weeks, then every other day. Nurse in constant attendance day and night; tracheotomy kept free of mucous by Sorenson pump; tube was removed in 24 hours, cleaned of blood and mucus, sterilized and replaced. Postoperative vomiting slight.

Feeding and Medication: First two days, tap water per rectum, a pint and one-half every four hours with 5 per cent glucose; next days with soda bicarbonate. For pain, codein phosphate, 1 gr. by hypodermic. Rectal medication of water and glucose stopped after second day. S.s. enema second day.

Nasal feeding began on second day postoperative; 1 oz. of warm water every half hour; later milk and water, 2-4 oz. Third day, chicken broth, 4 oz. Fourth day, eggnog. Fifth day, orange juice, coffee and milk. Cigarette drain was changed. Sixth day, ice cream soda. Plenty of mucus in tube. Eighth day, sat up for 10 minutes. Ninth day, walked to bathroom. Plenty of mucus in tube (tracheotomy), slightly bloody. Fourteenth day, nasal tube removed and liquid given by mouth did not come through wound. Twenty-fourth day, he went out on roof. Twenty-ninth day, he went for artificial larynx. Thirty-fifth day postoperative, he was discharged to clinic.

We used codein phosphate to control pain; fluid extract of belladonna leaves to control excessive mucus; and oil of thyme, with eucalyptus and menthol in liquid petrolatum sprayed on gauze over the tracheotomy tube to medicate the air.

Readmitted Aug. 8, 1931, and a tag of granulation tissue from mucosa in the tracheotomy wound was clamped, cut and touched with silver nitrate. Discharged Aug. 10, 1931, after applying adrenalin.

Readmitted Sept. 15, 1931, for excessive granulation tissue in wound about tracheotomy tube. Tissue removed and patient discharged Sept. 17, 1931.

Pathologist's Report: "Carcinoma of larynx."

After Events: Patient continued to use the artificial larynx for a year or so, then discarded it to a great extent as he was able to make himself understood without it. I have seen him many times since, the last time a year ago. Two months ago his cousin told me the patient was

doing well; two days ago one of his neighbors told me she saw him three weeks ago and he was working.

In closing, permit me to take this opportunity to pay tribute to one who not only allowed me to operate on this case, but guided me in the operative technique. To him, whose sound judgment, because of his long experience and pioneer work in this field, we all appreciate, to him is due greatest credit for the successful result in this case. I refer to none other than Dr. John D. Kernan.

20 Schermerhorn Street.

SYMPOSIUM: OTO-LARYNGOLOGIC CASE REPORTS.†

II.—MASTOID CONDITIONS.*

1. Facial Paralysis in Adult — Preoperative (Acute Mastoiditis).

2. Massive Osteoma of External Auditory Meatus with Osteoma of Temporal Bone.

3. Osteomyelitis of Zygoma Postoperative on Mastoid.

4. Acute Purulent Otitis Media, Acute Mastoiditis, Bilateral, Septicemia.

DR. RAYMOND J. GAFFNEY, New York.

In reviewing the case histories of the otological service of St. Vincent's Hospital for the past two years, one is impressed with the wealth of material which is found in this a general hospital. While the number of radical mastoidectomies is few, the acute cases are many, especially on the pediatric service. One is also impressed with the complications arising from the flare-up of chronic otorrheas. One case at present under our care came in with a high temperature and a running ear. After a few days of observation, during which time his temperature came down to normal and his ear complaint was negative, he suddenly presented the alarming picture of meningeal invasion with spinal fluid showing 7,000 cells and classical signs. Immediate operation showed a tremendous epidural abscess with marked necrosis of the tegmen. Very soon afterwards he developed symptoms of brain abscess with marked naming aphasia (lesion on the left side). Exploration of the temporosphenoidal lobe evacuated an abscess of over 100 cc. of pus. A Sirus drain was inserted. Today, after five weeks, the patient is well on the road to recovery. I feel that there is a tendency in chronic otorrheas to dismiss them with the instructions to the patient to instill drops and wait. There were perhaps in the past too many radical mastoidectomies, but today I wonder if we are doing too few. Would it not be

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well to arouse the public through proper channels to the danger of chronic running ears?

Case 1. Male, age 60 years, in good health, developed a mild upper respiratory infection. On the third day of illness he had a severe earache on his left side. The following day a myringotomy was done. The otoscopic picture was a bulging drum membrane with a large bleb in the external auditory meatus. The pus was small in amount. He went along nicely until the tenth day with scanty discharge, with little pain and no mastoid tenderness. His temperature ranged between 99-100°. On the tenth day I called to see him and found a well developed left facial paralysis of the peripheral type. Operation was performed two hours afterward. A very small sclerotic mastoid with arrested pneumatization was found with a scant amount of pus in the area of the antrum and near the tegmen. The rest of the mastoid cavity was free of pus. His postoperative course was uneventful, the wound healing promptly. On the third postoperative day the signs of paralysis of the face lessened and thereafter, from day to day, improvement was noted, until 12 days after operation all signs of the paralysis disappeared. This, I believe, was a rapid return of function.

While this picture has, no doubt, been seen occasionally by others, I feel that it is rare enough to report and that the treatment is prompt surgery on the mastoid. We know that young children suffer from facial paralysis preoperatively much more commonly than do adults. The question arises whether the pathology was due to pressure with dehiscence of bone, or to toxic origin. I believe that the former was the case. The pus was confined entirely to the antrum and immediate vicinity. Pressure on the facial nerve along the horizontal portion could account for the paralysis. Would recovery have been so prompt if of toxic origin, or does toxic origin always imply mechanical pressure? A simple mastoidectomy was sufficient treatment to restore function. We must, of course, be reasonably sure that the paralysis is the result of mastoid pathology as determined by history and signs. Dintenfass in the Pennsylvania Medical Journal, Aug., 1935, reports four cases of facial paralysis in middle ear disease. One developed a facial paralysis 24 hours after a catarrhal otitis, which completely disappeared in four days. Several years later the same sequence occurred. The second case, an adult, developed a facial paralysis a few hours after the initial invasion of the middle ear, with complete recovery in a week. The third patient, an infant with bilateral suppurative otitis media of four weeks' duration, developed a facial paralysis. A mastoidectomy was done and recovery of the facial paralysis took one month. The fourth patient, a child, age 12 years, developed paralysis three days after a severe influenzal otitis. A mastoidectomy was done four days later. Improvement of the paralysis was immediate, but complete recovery took six weeks. Should an early appearing facial paralysis demand mastoid surgery, or should we wait, and how long?

Case 2. Male child, age 11 years, undernourished, admitted to the hospital for a mild upper respiratory infection. The admission diagnosis on the ears was: "Right ear normal; left ear, a mild tubotympanitis;" temperature normal. Four days after admission the child complained of pain in the right ear and had a chill and vomited; temperature, 103° F. Upon examination a bulging ear was seen. The left ear was negative. The anterior cervical glands were swollen bilaterally; mild headache developed later in the day. A myringotomy was performed on the right side, releasing a serosanguineous fluid. The neurological report at this time showed a bilateral Kernig. Spinal tap showed clear fluid under moderately increased pressure. The following day the culture from the ear and from the blood showed streptococcus hemolyticus. The neurological report showed a stiff neck and bilateral Kernig. The following

day the symptoms were the same and 250 cc. of blood were given. The next day the Kernig was more marked; the neck very rigid. The otoscopic picture seemed to be clearing. Temperature at this point was 107°. The X-ray report at this time was: Right mastoid — cells of small pneumatic type with thin bony cortex; no evidence of pathology seen. (Remember that this is the pathological ear otoscopically.) Left mastoid — diffuse clouding of all cells with beginning softening in the region of the knee (this the negative ear otoscopically).

Prompted by the bacterial report and by the clinical picture presented, a double mastoidectomy was performed, followed at once by 200 cc. of blood. Next day the neurological signs were more pronounced; culture still showed streptococcus hemolyticus. The following day 160 cc. of blood were given. The report four days later still showed meningeal signs and positive blood culture. From that point onward, however, the child made rapid strides toward recovery, and 21 days from this point, both ears completely healed and the patient left the hospital cured. The operative findings were: Right side, small amount of pus in antral region, hemorrhagic elsewhere. A deliberate exposure of the sigmoid sinus was macroscopically normal. Left side, a coalescent type of pathology. The mastoid cells were easily exenterated.

The interesting points of this case were, on admission, the early advent of positive blood culture, the alarming meningeal symptoms and the peak of temperature, 107°. The ear, namely the left, which appeared negative throughout with an unruptured drum membrane, on operation proved to be the source of a very pronounced mastoiditis. It was on the strength of the X-ray report that the left mastoid was invaded. I believe we must once more give due credit to the transfusions for the happy termination of the case. Also, we were pleased that we explored the left ear along with the right and did not let the negative drum membrane influence us. X-ray diagnosis is, no doubt, helpful in some cases.

Lillie, in the Archives of Otolaryngology, Nov., 1933, reports 52 cases of otitic sepsis. The mortality was 13.5 per cent. In 33 the sepsis was preoperative, in 19 postoperative. Of the total of seven deaths, six occurred in the postoperative sepsis group, with only one where sepsis preceded operation. The reason for this discrepancy would be interesting to find. Gilroy Glass, in the English Journal of Laryngology and Otology, 1934, reports a mortality of 57 per cent in 63 cases. Seventy-one per cent of his series were postoperative blood infections. Does it not seem true that too early operation on the mastoid is not good surgery and that complications are not prevented thereby?

Case 3. Female, age 19 years, admitted to the hospital with a diagnosis of acute mastoiditis, right, was operated upon and found to have a very large pneumatic type of bone with suppuration very extensive. She was discharged on the twentieth day, the wound healing slowly. It was noted at operation that the zygomatic cells were very well developed and extended far forward. They were exenterated at the time of operation. Two days after discharge to the clinic she was readmitted with nausea, vomiting, chills and fever. Five days later a revision of the mastoid was done. Her temperature at this time was 105° F. The zygoma was completely exposed by a forward incision. Free pus was found between the periosteum and the zygoma. This bone was further inspected by biting away its exposed surface. A frank osteomyelitis was found, which also extended upward along the skull, involving the squama. All diseased bone was removed and the wound left wide open. Two days preoperatively the dental surgeon reported tenderness and swelling in the right parotid region and over the right temporal region; loss of function of the mandible with well marked trismus. Transfusion was done two days after operation with 300 cc. of blood. A plastic repair was done eight days postoperatively and in due course she was discharged from the hospital in good condition. While zygomatic mastoiditis is fairly common in

children, the involvement of the zygomatic bone itself is, I believe, not so common. This case had a happier termination than some of the bone infections of the skull.

Case 4. Female, age 43 years, consulted her physician for a recent loss of hearing. For the past 14 years she had noticed the gradual enlargement of a bony growth on the upper border of the squama, left side. For the last four months she claimed that she could not hear at all with her left ear. The ear was said to be discharging slightly for the past six weeks. She never complained before and stated that she was not conscious of any growth in her left ear, which statement, coming from a very intelligent woman, seems incredible when we view the extent of the mass. In her past history she said that a fatty tumor was removed from her jaw 12 years previously. Her physician referred her to me with a possible diagnosis of malignancy. On inspection, a smooth, shiny

Fig. 1. Case 4.

rounded mass presented itself just at the entrance of the external auditory meatus, and was readily seen without any speculum. On tapping, it presented a hard and solid feel. No discharge was noted, and the circumference of the canal was so completely sealed up that it seemed difficult to imagine any fluid passing by. On testing her hearing, she responded only to a shout.

Operation: One incision embraced both the area of the osteoma of the squama and that over the mastoid. The osteoma above was quickly reduced with a mallet and chisel. The pinna was retracted forward and the growth in the canal exposed. By careful manipulation the mass was loosened from a broad attachment to the posterior aspect of the bony canal and removed. A view of the tympanum was then possible, and due to the absence of any recognizable structures, and due to a foul necrotic mass, a radical mastoidectomy was done. Her further history was uneventful and she left the hospital on the sixth day following admission.

15 West 11th Street.

SYMPOSIUM: OTO-LARYNGOLOGIC CASE REPORTS.†

III.—PARAPHARYNGEAL INFECTIONS. CASE REPORTS.*

DR. EARL E. BAKER, New York.

By pharyngeal infection is meant an infection about the pharynx. The few minutes allotted to me will make it necessary for this report on the cases to be very brief.

Case 1. M. G., an Italian seaman, age 55 years, was admitted to St. Vincent's Hospital on Nov. 18, 1935, because of swelling below his jaw on the left side of his neck since 1914. This swelling would increase in size with intermittent attacks of fullness and soreness in the floor of the mouth that would last for a few days to a week, and would improve with improvement of the mouth condition. This last attack caused him to come to the hospital for treatment. Not affected by eating spicy or sour foods.

Past History: Passed stone in mouth following one of these attacks in 1915. Swelling was incised Aug. 16, 1935. No pus or stone was found at that time. Biopsy of tissue showed chronic inflammatory glandular tissue.

Examination: Temperature 100°. Firm, oval, fixed, slightly tender egg-sized tumor mass on left side of neck under mandible. Old scar of August operation. Mouth examination revealed moderate fullness and congestion over floor. Pus readily expressed from left Wharton's duct only. Probe passed into left Wharton's duct for 5 cm. revealed a grating feeling which was thought to be a calculus. X-ray revealed stone.

Operation: Usual aseptic preparation. Attempt was made to tunnel out duct after local application of 10 per cent cocain. Finally, it was necessary to use novocain infiltration. Duct more freely opened by cutting upward. Part of calculus removed in pieces, but could not remove the remainder because of poor co-operation on the part of the patient. Small incision made in neck anterior and below angle of jaw. No stone found.

The next day the patient passed a stony substance from mouth wound. X-ray later the same day showed no more evidence of stone.

Twelve days later a firm tumor mass was removed from the neck wound. Pathological examination showed this to be chronic reparative glandular tissue without acini.

Comment: Case is interesting because of its chronicity and because of the difficulty in removing the stone. Also the habit that stones have of expelling themselves after dislodgment.

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Case 2. E. T., radio salesman, age 28 years, was admitted to the hospital on April 28, 1935. Complaint: Pain to the left side of the neck below jaw, continuous and quite severe at times, for the past 24 hours, increased salivation for the past week, fullness in the floor of the mouth, sore throat.

Examination: Temperature, 103°; W. B. C., 18500; polys., 86 per cent. Tongue elevated, floor of the mouth swollen and congested. Pus expressed from left Wharton's Duct only. No palpable stone in floor of mouth. Tumor in neck egg-sized, oval, firm, adherent. Moderately tender but no fluctuation.

Operation: Usual surgical aseptic preparation. Anesthesia 2 per cent novocain. Three-inch incision over swelling, parallel and one-half inch below left mandible. Incision carried down, passing through platysma

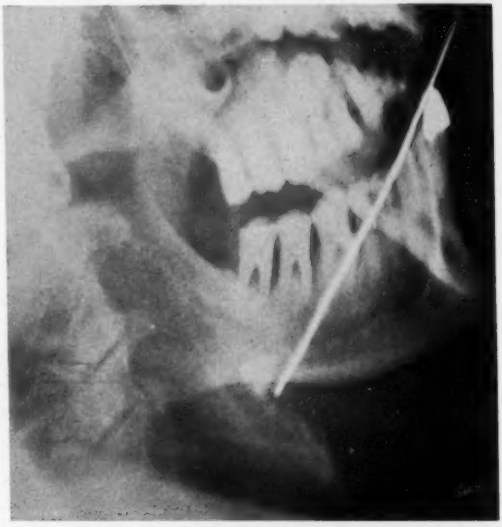

Fig. 1. Radiograph shows probe passed into left Wharton's duct. Stone is shown posterior and superior. Smaller stone at tips.

muscle, fibrous capsule into gland. Small amount of pus escaped but no stone found after a careful search. Stone passed next day after acute painful swelling in mouth, followed by considerable relief.

Comment: Operation dislodged the stone so that it readily passed into the mouth.

Case 3. G. V., an Italian woman, age 49 years, was brought to the hospital in an ambulance on Nov. 7, 1935, at 11 P. M. Temperature on admission, 99°. Complaint: Painful swallowing, lump in throat, and swelling in neck for last three days. Sensation of fullness and pains in the chest for the last four days. No history of foreign body in esophagus.

Examination: Well developed woman, age 49 years. Slight dyspnea. Markedly tender, diffuse swelling in lower part of left anterior triangle

of neck, no fluctuation. Throat markedly congested; moderate edema of glottis; slight trismus; temperature, 103°; W. B. C., 18400; polys., 82 per cent. Because of septic condition of patient, she was taken direct to operating room. Usual surgical preparation; 2 per cent novocain infiltration. A four-inch incision was made along anterior border of left sternomastoid muscle, beginning about one inch below angle of the jaw. After platysma and fascia was cut, incision carried down by blunt dissection to carotid sheath. No pus found, no venous thrombosis. Considerable friable tissue noted. Because of the rather severe dyspnea tracheotomy was done. Blood culture taken and blood for typing. X-rayed first day after admission. Patient looked septic. Temperature 104°, pulse full, rapid and bounding. Bronchiectic rales. Urine, 4 + albumin. X-ray showed a dense area in soft tissue occupying the region of the laryngopharynx with forward displacement of laryngeal structures. Malalignment in relation to 3-4-5 cervical segments, probably due to

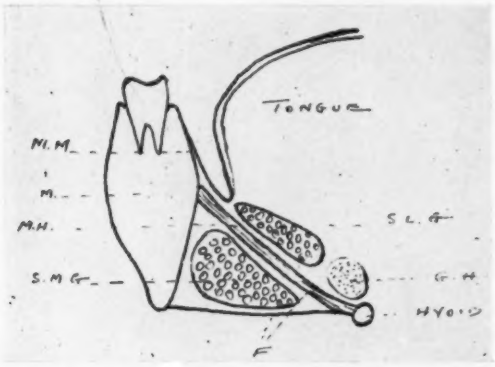

Fig. 2. (MM) Mucous membrane. (M) Mandible. (MH) Mylohyoid. (SLG) Sublingual gland. (GH) Geniohyoid muscle. (F) Fascia. This diagram indicates that while the submaxillary gland is separated from the mucosa of the floor of the mouth by muscle layer, it has a direct communication with the mouth cavity through its duct. The fascia acts as a barrier to downward passage of infection.

mass or abscess. Again taken to operating room (second day). Patient put in Rose position, cocain 10 per cent applied locally to retropharynx. Stab wound enlarged by forceps in this area by laryngoscope. Considerable bleeding, but no pus found. Second X-ray taken on the fourth day, but it did not show any change; however, the one taken eight days later showed marked improvement. Small amount of pus seen in wound on the seventh day. After a stormy convalescence, the condition finally improved; tracheotomy tube was removed on the twelfth day and she left the hospital in about one month. Blood culture was negative.

Comment: This is a case of retropharyngeal cellulitis, portal of entry probably the nasal pharynx. This stresses the importance of establishing an early airway in a case of this type. Tracheotomy was done after the operation, which is not the usual procedure. This emphasizes the importance of X-ray soft tissue detail technique as a diagnostic aid.

Case 4. N. R., male, Italian child, age 19 months, admitted to the hospital on May 10, 1935, with a temperature of 101°. Complaints: Harsh

breathing, difficult swallowing and swelling of neck, all of four days' duration. Temperature for the last 10 days.

Child had whooping cough five months ago.

Examination: Male child, age 19 months, not appearing acutely ill. No cyanosis or dyspnea. Ear drums dull. Banty-egg sized, fluctuating, swelling at the angle of the left jaw. Nasopharynx congested, slight purulent discharge in nose and throat. Soft doughy swelling in left retropharynx region. W. B. C., 28800; polys., g75 per cent; lymph, 21 per cent; urine, negative. Temperature when I first saw the child, next day, was 99° at 2 P. M. and child looked quite well. At 11 P. M. that night the temperature again went up to 101,° followed by a profuse discharge from the left ear. Temperature dropped and remained normal afterwards. X-rays taken five days later showed a moderate degree of anterior bulging of larynx and trachea, probably due to a retropharyngeal abscess.

Fig. 3. This radiograph shows dense area in soft tissue occupying the region of the laryngopharynx with forward displacement of the laryngeal structures. Malalignment in relationship of 3-4 and 5 cervical segments, probably due to cervical abscess.

Left mastoid showed generalized union in cells of antrum and mastoid regions, probably due to pressure of granulations or exudate. Right mastoid normal. The swelling in the neck and throat disappeared in 10 days, the left ear stopped discharging 16 days later, and the child was discharged shortly after in good condition.

Comment: Case is interesting from the standpoint that the child enjoyed good health with the pathology present. That the infection probably started from the nasopharynx, carried by lymph system into retropharynx, causing retropharyngeal abscess. From here burrowed into pharyngomaxillary space, into fascia free retromandibular area of parotid gland area into parotid gland, then into the fascia free external canal region into the left ear.

I believe this was a retropharyngeal abscess because of the ease in which the child could open his mouth, to the definite fullness in left lateral pharynx and to the mild degree of involvement of the tonsils.

ANATOMICAL CONSIDERATIONS.

It is evident that familiarity with the applied anatomy of the cervical fascia is indispensable to the solving of the problems arising from infection in the neck. According to Mosher, there are three fascial compartments in the upper neck which communicate more or less directly with the sheath of the great vessels. Those in the parotid submaxillary

Fig. 4. Shows improvements in soft tissue area occupying region of laryngopharynx, eight days later.

compartments which contain the respective glands and require no detailed description except to mention the fact that the retromandibular process of the parotid gland is devoid of fascia at its inner extremity where it lies in most intimate relationship with the pharyngomaxillary space. The submaxillary gland is separated from the mucosa of the floor of the mouth by a muscle layer, but does get a direct communication with the mouth cavity through its duct. The pharyn-

gomaxillary space is somewhat cone-shaped, the apex of the cone being downward with the continuous sheath of the great vessels and its base uppermost at the base of the skull. These three compartments can be considered hollow leaves and the carotid sheath the hollow stem.

The prevertebral fascia because of frequency of infection requires mention. This fascia encircles the vertebral column and the muscles attached to it. Infections have a tendency to

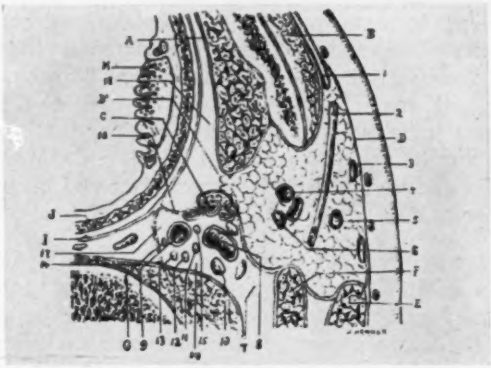

Fig. 5. This shows a section through the second vertebra and tonsil. (A) Internal pterygoid muscle. (B) Masseter muscle. (C) Styloid process with musculature. (D) Parotid gland with its fascia. (D) Absence of fascia over parotid. (E) Sternocleidomastoid muscle. (F) Digastric muscle. (G) Prevertebral muscles with prevertebral fascia. (H) Palatine tonsil. (I) Buccopharyngeal fascia. (J) Superior constrictor muscle. (1) Stenson's duct. (2) Fascial nerve. (3) Superficial parotid lymph gland. (4) External carotid artery. (5) External jugular vein. (6) Deep parotid lymph gland. (7) Fatty tissue in parapharyngeal lymph space. (8) Internal jugular vein. (9) Internal carotid artery. (10) Lateral pharyngeal lymph node. (11) Spinal accessory nerve. (12) Vagus nerve. (13) Superior cervical sympathetic. (14) Hypoglossal nerve. (15) Glossopharyngeal nerve. (16) Stylopharyngeal aponeurosis. (17) Fascial septum separating retropharyngeal from parapharyngeal space. (18) Prestyloid compartment. (19) Retrostyloid compartment. *(From Testut and Jacob.)*

travel rapidly downward. Pharyngomaxillary involvement is rare. If they start high, anteriorly, they usually extend more laterally, following the scaleni muscles. Infections which start laterally and anteriorly at the start will quickly reach the vascular sheath which crosses the scalenus obliquely. In other words, a true midline infection will extend into the posterior mediastinum, the lateral infections along the muscles under the clavicle into the costocoracoid region in the chest wall, into

the axilla and possibly into the posterior cervical triangle where it will become superficial.

The last important fascia is known as the visceral layer because it encircles the structures. In the lower part of the neck it is known as the pretracheal layers and surrounds the trachea and esophagus; it splits anteriorly to envelop the thyroid gland. In the upper part of the neck it is known as the buccopharyngeal fascia and covers the constrictor muscles and buccinator. This fascia is the seat of involvement when infections penetrate the hypopharynx and esophagus.

According to case reports of fascial involvement, the pharyngomaxillary space, either alone or with others, is the one most frequently involved. In a series of 24 cases reported by Beck, all requiring operation but two, the following percentages were noted: Pharyngomaxillary, 55 per cent; submaxillary, 20 per cent; cervical lymph node and prevertebral fascia and carotid sheath, 3 per cent; and buccopharyngeal, 2 per cent.

There are two types of cervical infection from a pathological as well as a clinical standpoint: A phlegmonous type, usually coming on from four to seven days after initial infection, in which the local signs of inflammation are predominant; the other, rare vascular type in which these are absent or subordinate, though symptoms from the start being suggestive of systemic involvement.

Phlegmonous Form: This is primarily a cellulitis of the loose tissue within the prestyloid compartment to which it probably remains confined in uncomplicated cases. Abscess formation takes place in the majority of cases. The diagnosis rests on three cardinal signs, in their order of appearance: Trismus, fever and swelling of the neck on the affected side. The trismus, and soon marked, is generally the earliest of the symptoms. Temperature usually, 101-102° F. May be at first only in the late afternoon, but in a few days the patient often presents the typical picture of sepsis with chills preceding the sweats. A temperature over 102° F. is suggestive of an additional complication. The tender swelling of the upper part of the neck is noted before the end of the week. Extension downward does not take place to any marked degree for several days in most cases; may spread over mandible

and face. Pain in throat often referred to ears. Some headache may portend a basal meningitis. Edema of fauces usually involves uvula early, then the pillars and palate, giving the appearance of peritonsillar abscess, which is one of the late signs. Swelling of lateral pharyngeal wall may be with or without change in color and the uvula may point horizontally to the opposite side. The tonsil looks normal, thus ruling out peritonsillar abscess.

Complications: If the case does not recover in two to three weeks, the following complications are possible: Mediastinitis, jugular thrombosis, edema of larynx, Ludwig's angina, fatal hemorrhage, osteomyelitis, pneumonia, erysipelas, vagus involvement, meningitis, parotid abscess and septicopyemia.

Vascular Type: The symptoms are those of septicemia, thrombosis or embolism. Primarily, it may be conceived as an infective process, either involving or close to one or more of the venous channels in the pharyngomaxillary space.

Because of the common occurrence of infections of dental origin, a few words will be said about the so-called cases of Ludwig's angina. These are of two kinds, usually caused by the streptococcus. Origin of the nondental type is obscure, probably of lymph gland origin in the floor of the mouth. Both manifest themselves similarly. In the dental cases there is swelling and induration of the floor of the mouth. The infection rarely remains confined to the submaxillary space but extends into the parotid space, pharyngomaxillary space, or both, and downward below these into the lower part of the neck along the vascular sheath. The applied fascial anatomy is borne out clinically. Those of dental etiology yield promptly to submaxillary drainage and can be usually given a good prognosis. In these the trismus is extreme and the floor of the mouth is usually involved. Those not of dental origin are more dangerous and will not show the mouth swelling and trismus unless the pharyngomaxillary space is involved. These cases should be drained early and if sepsis continues and chills occur the carotid sheath should be exposed. Positive blood culture indicates immediate ligation of jugular vein to prevent distant thrombophlebitis. If marked sepsis, particularly with chills, indicates ligation.

In conclusion, a few words will be said about surgical approach for drainage. The skin incision must be ample.

1. Retropharyngeal abscess: *a.* Direct incision of pharyngeal wall through the mouth. *b.* Dean's incision along anterior border of sternomastoid lateral to the larynx, reaching the visceral fascia and following this around to the prevertebral fascia, thrusting forceps or blunt instrument into abscess cavity. This incision minimizes manipulation of carotid sheath and avoids the underlying sympathetic nerve and may avoid tracheotomy where mechanical respiratory obstruction is present.

2. Pharyngomaxillary, parotid and submaxillary space infection: *a.* Skin incision of secondary importance, providing it is long enough to give a wide exposure. Mosher advises T-shaped incision which gives a wide, flat exposure because veins that will flood the field can be easily tied. The crossbar of the T runs parallel with the border of the jaw and close to it. Its object is to expose the submaxillary gland. After the facial vein is tied and cut, the lower border of the gland is elevated near its lateral extremity, the finger is inserted beneath it and carried backward and upward along the deep layer of its fascia until the stylomandibular ligament is felt beneath the angle of the jaw. It is then in a position to enter any of the three uppermost deep fascial spaces. With this ligament located, the finger is carried upward along it until the styloid process is felt. By carrying the finger upward and external to the styloid process to the base of the skull, it has entered the pharyngomaxillary space. By carrying the finger forward from the ligament on the inner side of the jaw, it gets into relationship with the side of the tongue, floor of the mouth and region of the tonsil. This area can be felt through the pharynx. The vascular sheath lies deep to the styloid process and is, therefore, drained at that point by this maneuver.

3. Carotid sheath and visceral fascia infection: *a.* Incision along anterior border of sternomastoid as for jugular resection.

4. Posterior triangle infection: *a.* Incision along or behind posterior border of sternomastoid.

A word about bacteriology from case series indicates that the hemolytic streptococcus causes over 50 per cent of neck infections.

Another interesting analysis of the etiology as shown by Mosher is that the greatest single cause of these infections is "in or about the tonsils." Forty-five per cent, next in frequency, comes the pharynx.

A word at this time will be said about the treatment of the pharyngomaxillary compartment and it will depend naturally upon the part involved, whether it is in the prestyloid space or retrostyloid space and the possible complications that may exist when the case is seen. But essentially the treatment is expectant because of the tendency of the prestyloid infections to localize. This may be aided by hot gargles, electric pads on neck and position of the patient with infected side up, the patient lying on the opposite side. As soon as localization is noted early surgery should be done, either by way of the mouth or externally.

150 East 90th Street.

SYMPOSIUM: OTO-LARYNGOLOGIC CASE REPORTS.†

IV.—ESOPHAGEAL CONDITIONS.*

1. Imperforate Esophagus.

2. Carcinoma of Esophagus — Two Cases.

3. Cardiospasm Mistaken for Carcinoma of Esophagus.

DR. ANTHONY NIGRO, New York.

The following list of cases is presented to show some of the pitfalls that we can get into if we are not careful in the study of esophageal cases. First, the history of any particular case should be stressed. Fluoroscopy with an opaque mixture should be done, followed by X-rays, with careful esophagoscopy and biopsy whenever necessary. An early diagnosis is important in the treatment of any case if we are to get any result from any surgical interference.

Case 1. Baby I. Imperforate esophagus, with fistulous tract between trachea and esophagus.

Present History: Female infant, born Oct. 21, 1934. History of inability to retain fluids by mouth immediately after birth. Clinical diagnosis was made of a possible congenital anomaly of the esophagus. An esophagoscopy was requested by the pediatric service and was done the day after birth. Esophagoscopy revealed a blind pouch, five inches from the upper jaw, at the junction of the middle and upper thirds of the esophagus. The larynx was patent. This was confirmed by X-rays taken immediately after the barium had been introduced into the pouch. X-rays also revealed the presence of air in the stomach. This was of considerable diagnostic importance, indicating some connection between the respiratory tract and the stomach. On the basis of these findings, a gastrostomy was done on the same day, under local anesthesia.

Further esophagoscopy was done with lipiodol and in the course of the fluoroscopic study, the lipiodol spilled from the esophageal pouch and entered the trachea via the larynx and appeared in a short moment in both lungs, lower end of patent esophagus and in stomach. This confirmed the existence of a fistulous tract between the trachea and esophagus. The X-rays readily confirmed the above findings. The baby was kept alive for over 40 days by judicious use of gastric feedings, and in such amounts that none would get into the esophagus and into the lungs. A bronchoscopy should have been done, but it was not done for nonprofessional reasons.

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The interesting part of the case was that a small esophagoscope, introduced from above in the esophageal pouch, and one introduced from the gastrostomy wound during a retrograde esophagoscopy, showed that the ends of the two esophagoscopes were not more than 2 cm. apart. The intervening tissue was made up of muscle and connective tissue, and with some pressure the ends of the two esophagoscopes would meet. Would it be possible for any surgical intervention in this case? Primarily, the tracheoesophageal tract should be closed first so as not to allow any food to spill into the lungs and cause a bronchopneumonia, which is the usual complication in these cases. The baby died on Dec. 4, 1935.

Postmortem: Trachea was removed, together with larynx, pharynx, esophagus and stomach. Above the carina and on the posterior wall, there was a small opening. A probe introduced into this entered the stomach. Water introduced also flowed into the stomach. The esophagus and pharynx externally appeared continuous. Up to the level of the carina of the trachea, the esophagus was wide, and then rather abruptly narrowed down and remained so, as far as the stomach. Water introduced into the upper esophagus reached the narrowed portion and proceeded no further. Pressure had no effect in forcing the fluid into the narrowed lower esophagus. We thus had this tube divided into two noncommunicating tracts, although their musculature was continuous There was a blind upper tract extending to the level of the carina of the trachea and the lower esophagus, whose lumen was continuous on one hand with the trachea above and the stomach below.

Case 2. Diagnosis: Carcinoma of the cardiac end of the esophagus. A. D., age 59 years, was admitted on Sept. 11, 1934, and discharged Nov. 12, 1934, with complaint of dysphagia of eight weeks' duration; loss of 29 pounds in five weeks; hematemesis twice, about half-pint of blood.

Present history dated back about eight weeks, at which time he had a choking spell following the ingestion of a small piece of meat. He gave a previous history of pain in the anterior chest and feeling that something was growing there. He had been able to swallow liquid food occasions following the ingestion of solid food. Fluoroscopy on Sept. 20, only for the past eight weeks. The patient had hemorrhages on two 1934, showed the following: About two inches above the diaphragm there was a constant narrowing of the lumen, above which there was a dilatation of the esophagus. The barium trickled through this constriction, which was approximately two and one-half to three inches in length. X-ray films confirmed the fluoroscopic findings. The characteristics of this deformity were definitely in favor of carcinoma of the cardiac end of the esophagus. An esophagoscopy was done. The tube met an obstruction at the cardiac end of the esophagus. No ulceration was present. A biopsy was taken.

Pathological Diagnosis: Squamous cell carcinoma No. 2.

Jejunostomy was done on Oct. 11. Following the esophagoscopy, the patient began to complain of difficulty in swallowing in the upper part of the esophagus, with a slight elevation of temperature. An indirect laryngoscopy on Oct. 3 showed the following: Obliteration of the right pyriform fossa; fullness of lateral and posterior right pharyngeal wall; slight compression of larynx from right to left; redness of tissues over the area of fullness. External examination showed tenderness along the anterior border of the sternocleidomastoid muscle on the right side at the level of the cricoid cartilage, with a slight fullness, brawniness and some crepitus in this region. X-rays revealed a surgical emphysema. Evidently, this seemed to be a secondary infection of the pyriform fossa with surgical emphysema caused by trauma during the course of the esophagoscopy. A spontaneous rupture occurred, and no operative procedure was resorted to. This is the danger in every esophagoscopy. The patient left the hospital and later died in Bellevue Hospital.

Case 3. DeS. S., age 64 years, admitted Aug. 16, 1935.

Chief Complaint: Difficulty in swallowing, dating back one month.

Present History: Patient had one attack of dysphagia six months ago, which came on suddenly when he was swallowing a mouthful of meat. This attack lasted only during that meal. He regurgitated the food, but thereafter he was able to eat everything without difficulty. One month ago he suffered an attack identical with the first, and continued to have such attacks intermittently ever since. At times he was able to swallow solid food, and at times he could only take water. There was no actual vomiting, nausea, or hoarseness; no hemoptysis or loss in weight. Good appetite. At the same time a swelling appeared over the right submaxillary and parotid region. This swelling persisted.

Previous history negative. On Aug. 17, 1935, examination of the esophagus after the ingestion of an opaque mixture showed an area of narrowing in the lumen of the tube above and below the clavicular level, with an area of dilatation in the first part of the esophagus. The appearance of the area of organic narrowing was suggestive of carcinomatous changes. A fluoroscopic examination of the esophagus during the ingestion of an opaque paste, showed an area of irregular constriction in the upper third of the esophagus, but no appreciable delay in the passage of the paste.

The first esophagoscopy done in July, 1935, was not satisfactory, and patient was asked to return within one week so that esophagoscopy could be repeated. Evidently there was a pathological condition in this region because when the patient returned after two months, a biopsy taken from a friable mass in the region of the cricopharyngeus was reported as squamous cell carcinoma. Gastrostomy was done on Oct. 8, 1935.

This was a case of carcinoma at the region of the cricoid, diagnosed clinically by fluoroscopy, X-ray and biopsy reports. The glands in the cervical region were found to be cancerous on biopsy. Postmortem was done.

In these cases a gastrostomy should be done early on account of dehydration acidosis and contraction of the stomach. If any surgical procedure is to be done, it should be done early, before any gland metastases occur, with possible resection of the upper end of the esophagus. The exposure of this region is simple through the anterior border of the sternocleidomastoid muscle. There was no paralysis of the recurrent laryngeal nerve, even though there was an extensive involvement of the esophagus, trachea, mediastinal and upper cervical glands.

Case 4. Diagnosis: Cardiospasm. J. C., age 65 years, admitted July 31, 1935.

History: Difficulty in swallowing for past four or five years, patient complaining of difficulty in swallowing solid or liquid food. This was accompanied by severe coughing and the spitting up of any substance ingested. His diet was mostly soft food. On July 30, 1935, the patient attempted to eat some meat which precipitated a severe attack of coughing and the meat was stuck in the throat.

A barium fluoroscopy of the esophagus was done, and an area of deformity involving the middle third of the esophagus was seen. The barium trickled down the esophagus along the side of the obstruction. No evidence of obstruction to the descent of the barium mixture was noted during these operations. The area of deformity suggested an infiltrated lesion. An esophagoscopy was done, and the esophagoscope entered without difficulty up to about 30 cm. from the margin of the upper teeth, where an obstruction was met. There seemed to be a piling up of mucous membrane at this region, and a fungating mass appeared in situ. A biopsy

was taken. In view of the clinical, X-ray and esophageal findings, a gastrostomy was done before the pathological report was received; however, the pathological diagnosis was "hyperkeratosis of the mucous membrane." Esophagoscopy was repeated. There was no obstructive lesion at the place where it was originally encountered, but instead, an obstruction was met at the cardiac end of the esophagus. The lumen of the esophagus appeared puckered and resistant to the passage of the esophagoscope. The biopsy report was the same as the previous one. The diagnosis of carcinoma was ruled out and a diagnosis of cardiospasm was made.

Apparently the findings of the previous X-ray and fluoroscopic studies were misinterpreted. The fungating mass was the bolus of food stuck in the esophagus. What appeared as an area of deficiency in the wall of the esophagus was really the barium trickling down the side of the bolus of food. Succeeding X-ray, fluoroscopy and esophagoscopy studies confirmed the above findings. The gastrostomy wound was utilized to dilate the cardiospasm. The patient was allowed to swallow a string which was grasped in the stomach through the gastrostomy wound, and a Tucker retrograde, esophageal dilator beginning with No. 20 French dilator until No. 28 dilator was used. The symptoms progressively improved. The patient is able to eat practically everything. The gastrostomy wound was closed.

St. Vincent's Hospital.

SYMPOSIUM: OTO-LARYNGOLOGIC CASE REPORTS.†

V.—MALIGNANCIES OF THE EAR, NOSE AND THROAT.*

1. Epithelioma (?) of Ear.

2. Lymphosarcoma of Nasopharynx — Two Cases.

3. Carcinoma of Nasal Sinuses — Two Cases.

4. Lymphosarcoma of Tonsil — Two Cases.

5. Epithelioma of Cheek.

6. Carcinoma of Larynx — Three Cases.

DR. EDWARD MARKEY PULLEN and
DR. JOHN M. LORE, New York.

To present an exhaustive or even an analytical study of malignancies of the ear, nose and throat in the allotted time, is well nigh impossible; however, a critical analysis of the salient features may serve a useful purpose.

It is indeed surprising when one realizes with what frequency malignant lesions are encountered in our specialty and gratifying indeed to note the good results obtained when an early diagnosis is made and proper treatment instituted. Just a few of the St. Vincent's Hospital cases are reported, illustrating the need for constant alertness and importance of instituting early and extensive treatment.

Case 1. Malignancy of the nasopharynx.

Mrs. M. S., age 50 years, had been under our care for a chronic purulent sinusitis for three years. Careful examination during that period had never shown any suspicious mass in the nasopharynx. On Sept. 18, 1931, she first complained of fullness of the right ear. Examination of the ear showed marked retraction of the drum, but no sign of inflammation. Inflation was attempted but was unsuccessful; therefore, a posterior rhinoscopy was done, and much to our surprise we found a moderately sized mass in the right nasopharynx pressing against the Eustachian tube orifice. Biopsy proved this mass to be a lymphosarcoma. Radium treatment was at once instituted. In a short time the mass disappeared and to date has not returned.

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Oct. 26, 1931. 15 mc.—10-1.5 mc. gold implants—four implants inserted into growth in nasopharynx via nose; six implants inserted into growth in nasopharynx via mouth—1995 mc. hours.

Nov. 2. 150 mg.—3 cm. distance—to right neck—22 hours; 3300 mg. hours.

Nov. 17. 100 mg.—2-50 mg. tubes—to right nares—2 hours; 200 mg. hours.

Nov. 17. 12 mc.—6-2 mc. gold implants—four implants inserted into nasopharynx, two implants inserted into nose; 1596 mc. hours.

Dec. 3. 150 mg.—3 cm. distance—each side of neck—18 hours; 2700 mg. hours.

Case 2. Mr. T. C., age 20 years, was first seen Nov. 10, 1926. Briefly, the history and findings from his first visit to the time a diagnosis of lymphorsarcoma was made, are:

1926. Was treated for O. M. P. C. (residua) left. No history of bleeding.

1928. For past several months has been bleeding from the back of his nose.

1930. Found a ruptured blood vessel, but no mass was identified as such.

1931. Removed his tonsils; no adenoids or mass found in nasopharynx.

Jan. 18, 1933. Severe bleeding from right nostril and postnasally.

Jan. 31. Mass in nasopharynx (right) noticed for the first time.

Feb. 13. Biopsy established diagnosis of lymphosarcoma.

Immediately after the diagnosis was established, radium treatment was instituted. When last examined on April 24, 1935, there was no evidence of a growth in the nasopharynx.

Feb. 15, 1933. 3 mc.—2-1.5 mc. gold implants—inserted into growth; 399 mc. hours.

Feb. 15. 100 mg.—2-50 mg. tubes—left side of neck—20 hours; 2000 mg. hours.

Feb. 15. 100 mg.—2-50 mg. tubes—right side of neck—20 hours; 2000 mg. hours.

Feb. 24. 100 mg.—2-50 mg. tubes—one tube each nares—two hours; 200 mg. hours.

Feb. 24. 100 mg.—2-50 mg. tubes—to soft palate—two hours; 200 mg. hours.

Feb. 24. 200 mg.—4-50 mg. tubes—100 mg. each neck—20 hours; 4000 mg. hours.

March 16. 100 mg.—2-50 mg. tubes—to soft palate—three hours; 300 mg. hours.

March 16. 50 mg.—tube—to left nares—three hours; 150 mg. hours.

May 10. 150 mg.—3-50 mg. tubes—to left cheek—four hours; 600 mg. hours.

May 10. 150 mg.—3-50 mg. tubes—to right cheek—four hours; 600 mg. hours.

In both these cases complete examination and X-rays showed no evidence of any metastasis before treatment was begun.

These two cases with the lesion located in the same region presented different symptoms. In the case of the woman, the chief complaint was fullness of the right ear. This symptom, while not significant, per se, assumed greater importance when it was found that the right ear could not be inflated. It immediately directed attention to the Eustachian tube orifice. Postrhinoscopic examination, as stated above, revealed a mass in the right nasopharynx closing off by pressure the Eustachian tube orifice. A biopsy at once revealed the true nature of the growth, making early treatment possible. Recent examination shows no signs of recurrence.

The other case (T. C.) is perhaps more interesting and instructive. Here the chief complaint was epistaxis. Nasal examination showed the bleeding to be coming from the nasopharynx and not from the usual place on the septum. No mass was recognized at that time. The bleeding having been controlled, the next step should have been a careful examination of the entire nose and nasopharynx. There is no doubt but that this procedure would have revealed the mass in the nasopharynx. But the patient failed to return for a check-up and had it not been for a recurrence of the bleeding, the growth might not have been detected until considerably later and perhaps too late for treatment. This time, however, a determined effort was made to examine the nasopharynx. For this purpose a self-retaining soft palate retractor was used after proper cocainization of this region. Using a laryngeal mirror (No. 5) it was but a simple matter to see the large mass in the right nasopharynx. A clinical diagnosis of lymphosarcoma was made, which was soon confirmed by biopsy, performed at the hospital. Radium treatment by Dr. G. A. Robinson was at once begun with the result as above stated.

Case 4. Malignancy of the sinuses.

Mr. W. O'S., age 35 years, was first seen by us on March 5, 1935. The history given was that about Dec. 1, 1934, his right eye began tearing. About the same time he noticed nasal obstruction (right). About Jan. 10, 1935, he first noticed some exophthalmos of the right eye. He also had slight pain. He consulted an ophthalmologist who treated him for dacryocystitis. Shortly before he was referred to us, the ophthalmologist had found what he called a polyp in his right nostril and attempted to remove it, but was unsuccessful.

When first examined by us, he had a marked exophthalmos but good vision, considerable redness at the inner canthus and pus coming from the lacrymal sac. He had diplopia. His right nostril was filled with brownish pus. After aspiration a mass was seen in the ethmoid region. Transillumination of his sinuses showed the right side to be 4+. A clinical diagnosis of malignancy was made and a biopsy performed at once. The tissue examination report (Dr. Fraser) was squamous cell carcinoma, Grade IV. Operation was suggested, and on March 11, 1935, a Caldwell-Luc and transantral ethmoidectomy (under local anesthesia) was performed. A malignant mass was found involving the floor of the orbit and the nasal antral wall, and extending into the ethmoids. Removal of the orbit and orbital contents was suggested but refused. So it was felt at the time of operation that the orbit was involved. Surgery was followed by radium treatment.

March 20, 1935. 100 mg.—2-50 mg. tubes—right antrum—22 hours; 2200 mg. hours.

March 27. 50 mg.—tubes—right antrum—18 hours; 900 mg. hours.

The improvement at first was marked. The exophthalmos almost disappeared, but soon returned and became quite marked, so that the

orbital contents were removed. Malignancy was found here. He did well for a while but soon there was necrosis of the bones of the orbit which eventually was followed by a profuse hemorrhage and signs of meningitis. Death occurred on Aug. 13, 1935.

Case 5. Malignancy of the antrum. Mrs. K. S., age 67 years, was first seen on Sept. 7, 1935. Her history was that for several months she had had occasional pains on the right side of her face and that for the past five weeks her symptoms had been acute. She also had considerable brownish discharge from the nose. Transillumination showed the right side to be 4+. She had at this time some fullness and induration in the anterior antral wall. It was suggested to her that her antrum be washed. On introducing the trocar for the lavage, there was severe bleeding. No bone (antral wall) was encountered, the trocar entering at once into the antrum. The trocar was at once withdrawn because an erosion of the antral wall was suspected. The right side of the nose was shrunk down and re-examined, whereupon a peculiar mass was seen high up in the ethmoid region. A biopsy was performed, but strangely enough the report came back "fibrous tissue." In spite of this, a clinical diagnosis of malignancy was made, and to confirm this an intranasal antrotomy was performed at the hospital. Examination of tissue removed from the interior of the antrum showed squamous cell carcinoma, Grade III (Dr. Fraser). On Sept. 19, 1935, a Caldwell-Luc and transantral ethmoid operation was performed. At the time of operation the anterior, outer, upper and inner walls of the antrum were eroded. A large mass filled the entire antrum. This was easily removed. No extension into the ethmoids was found. On Sept. 24, 1935, radium treatment was started.

Sept. 24. 100 mg.—2-50 mg. tubes—right antrum—30 hours; 3000 mg. hours.

Oct. 9. 80 mg.—2-40 mg. tubes—right antrum—20 hours; 1600 mg. hours.

Nov. 9. 50 mg.—tube—right ethmoid—20 hours; 1000 mg. hours.

To date she is much improved. No clinical evidence of malignancy has been noted, but she is being carefully watched.

These two cases illustrate above all else the importance of the time element. In the first, the diagnosis was not made until extension had taken place into the orbit. The second of these cases is very instructive. A clinical diagnosis of malignancy was made because of the symptoms character of the discharge, and, above all, because of the deficiency in the nasoantral wall and severe bleeding discovered at the time of the antral lavage. A first negative biopsy did not settle the diagnosis but made it imperative to resort to other measures to establish a definite diagnosis. As noted above, this was done, making early treatment possible. This case is by no means "out of the woods," but we have hopes of an ultimate cure.

Case 6. Epithelioma of the cheek. On Oct. 9, 1935, Mr. A. J., age 55 years, came in for an ear inflation and casually mentioned the fact that he had been recently examined by an insurance doctor, who told him he had leukoplakia of the mucous membrane of the mouth. Immediately we inspected his mouth and found a large hard mass about 1.5 cm. in diameter on the inner surface of the left cheek. On the inner surface of the right cheek there was an area of leukoplakia, irregular in outline, about 2 cm. in diameter. He had smaller similar areas on the side of his tongue. The mass on the left cheek was diagnosed as malignant and biopsy advised at once. This was done. In performing it, a large tonsil punch was used to grasp the mass, but when it was found that the mass was too hard for the punch, it was dissected out by means of a scissors. After the removal of the mass, the edges of the wound were electro-

coagulated. The biopsy report was epithelioma. A more extensive resection was advised and on Oct. 16, 1935, he was operated upon at St. Vincent's Hospital. An area about 1 cm. beyond the first removal was dissected but showed no malignancy. Since then, the area in the right cheek has been destroyed by electrocoagulation. The areas on his tongue have disappeared as a result of stopping his smoking.

Incidentally, it may be mentioned that he had known of these "white spots" on his cheeks for over 20 years and that he was a heavy smoker of strong cigars.

It seems incredible that such an obvious lesion should have been overlooked for so long. This case also stresses the importance of complete examination.

Case 7. Lymphosarcoma of the tonsil. E. B., a woman, age 47 years, was referred on May 24, 1935, as a result of a consciousness of a mass in the throat on the right side for the past two weeks. Examination showed a large right tonsil with plugged crypts of a pale bluish color. No other involvement was noted. The growth was removed on June 5, 1935, and verified the diagnosis of lymphosarcoma of the tonsil. Removal was followed by X-ray therapy and to the present time no recurrence or glandular involvement has been noted.

June 15, 1935. 200 mg.—5 cm. 24 hours—right side of neck—4800 mg. hours.

June 17. 200 mg.—3 cm. 18 hours—right side of neck—3600 mg. hours.

July 31. 300 mg.—3 cm. 20 hours—right side of neck—6000 mg. hours.

Dec. 2. 350 mg.—3 cm. 20 hours—right side of neck—7200 mg. hours.

Case 8. Lymphosarcoma of right tonsil. Mr. B. N., age 42 years, on Oct. 8, 1925, complained of a soreness in the region of the right tonsil and hoarseness for two days. Three weeks preceding this he was supposed to have had diphtheria. He had no rash but had a cough recently.

Examination revealed a deep ulcer of the right tonsil with some overgrowth of tissue. Wassermann and smears for Vincents' angina were negative, but there were some palpable glands on that side.

Six days later the tonsil was removed and found to be lymphosarcoma. This removal was followed by X-ray treatments which caused a severe reaction, but up to the present there has been no recurrence, and the removal was 10 years ago.

These two cases show good results, due to the examination of the tissue and being followed by the radiation.

Case 9. Carcinoma of the left vocal cord. Mr. V. D. D., age 65 years, gave as his chief complaint hoarseness for four or five months. Examination showed an irregular hard mass involving the entire cord, extending subglottically. There was some fixation of the left vocal cord. Biopsy showed carcinoma.

Laryngectomy was advised but was refused. A laryngofissure operation was done on July 10, 1931. This was extensive, going down to the cricoid; and the vocal process was removed. The after-results were good.

On Oct. 24, 1932, a gland appeared on the left side of the neck. This was removed on Nov. 2 and found to be a carcinoma of the gland in the carotid sheath, attached to the internal jugular vein; so it was removed with the vein. There was an uneventful recovery and the patient has been well up to the present.

Case 10. Carcinoma of the vocal cord. Mrs. K., age 47 years, was first seen on April 11, 1932, with the chief complaint of hoarseness for 15 months. Examination revealed a small mass on the middle third of the vocal cord. There was no impairment of motion, no inflammation and no glandular involvement. X-rays and Wassermann were negative.

Removal of the mass was advised and refused. The patient consulted another doctor who called it a benign growth. On May 25, 1932, the patient returned and an impairment in movement was noted. Diagnosis of possible carcinoma was made. Biopsy was done on June 1, 1932, resulting in the diagnosis of carcinoma, Grade II+. The patient was operated on soon after. Laryngofissure of the Lore technique was done. She had an uneventful recovery and up to the present has a good voice.

In the first case the malignancy was extensive and would ordinarily require a laryngectomy. This having been refused, a laryngofissure was done, which was most extensive.

The next case looked clinically benign. This stresses the importance of biopsy. As a matter of fact, nearly any early malignancy may have a benign appearance as evidenced by the absence of infiltration and the absence of immobility.

Case 11. Carcinoma of the external auditory canal. Mrs. C. F., age 47 years, on Nov. 8, 1928, was referred with the history of a recurrent granuloma of the left external auditory canal. One report had come back epithelioma. This was followed by radium treatment. When a recurrence of the mass showed, another specimen was taken which was found to be a sarcoma. This time, on Dec. 15, 1928, an extensive simple mastoidectomy was done and followed by radiation.

There was an uneventful recovery and the patient has been well up to the present. Recent X-rays (Dec., 1935) show no evidence of malignancy involving the mastoid area.

IMPACTED DENTAL PLATE IN LARYNX; REMOVAL BY SUSPENSION.

DR. E. LEE MYERS, St. Louis, Mo.

In a somewhat desultory perusal of the literature one is struck by the frequency with which dental plates become lodged in the esophagus; however, there is a paucity of cases where a plate of the size herein to be described has entered the larynx. The following case report has proved very interesting to me because of its rareness:

Case Report: E. C., male, age 42 years. While on a fishing trip June 3 he had an attack of grand mal and upon recovering consciousness, the group with whom he was fishing noticed that he was hoarse; however, five days later when he presented himself to his local physician, Dr. R. Graham Fish, of Paris, Tenn., he was slightly dyspneic with his hoarseness. The patient volunteered the information that he was missing his partial upper plate, which was originally a two-tooth vulcanite denture which extended from the upper right side of his mouth to the left. The tooth which was on the left had broken off but the patient had worn the bridge for considerable time without difficulty as it held itself in place very well by suction. A fluoroscopic examination showed the plate to be in a sagittal position in the larynx, but by indirect examination by Dr. Fish no evidence could be seen. I confirmed this examination late that evening by direct inspection. X-rays were made which proved the presence of the foreign body in the larynx.

Operation: June 8, 1936, McSwain Clinic, Paris, Tenn. The larynx was exposed by Jackson's laryngeal speculum; the foreign body was seen as a reddish mass in the anterior commissure but it was difficult to release same because the patient was struggling and considerably dyspneic; a second trial proved also to be a failure. At this time the patient was cyanotic; he was given oxygen and after a few minutes' delay a local tracheotomy was done and an attempt made to remove the intruder through the tracheotomy wound. The object was

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grasped but could not be delivered, possibly due to the width of the base of the dental plate which had toggled. Conse-

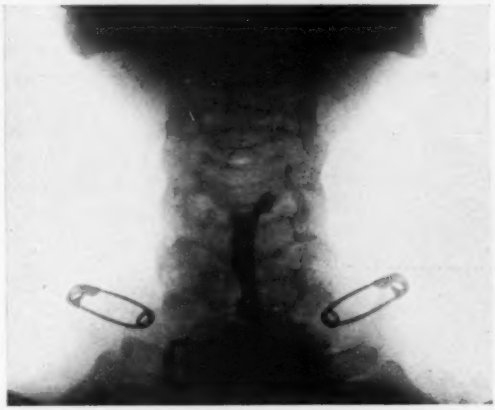

Fig. 1. Anterior-posterior view. Tracheotomy tube in position. Dental plate in subglottic larynx and trachea.

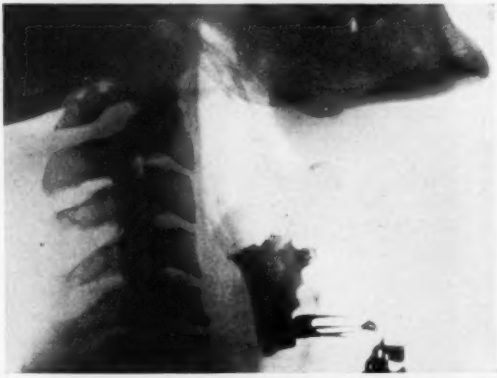

Fig. 2. Lateral view. Dental plate base up, tooth anterior acting as hook in anterior commissure, sharp end of base also holding posteriorly because of wedge-like shape.

quently, inasmuch as the disturbance of breathing had been satisfactorily handled, the patient was returned to his bed for a subsequent trial when able to visit St. Louis.

Second Operative Procedure: Jewish Hospital, June 26. Avertin anesthesia enhanced with ether through the tracheotomy opening. The patient was suspended with the Killian

Fig. 3. Dental plate removed; tracheotomy tube in place.

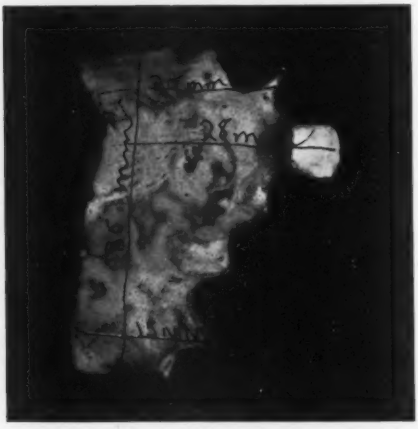

Fig. 4. Dental plate enlarged one-quarter.

suspension apparatus (Lynch modification). Here one found considerable edema of the left false cord, which, after being pressed towards the left, showed a reddish mass having a

convexity which practically occluded the larynx. Grasping the convex portion with the Irwin Moore alligator rat-tooth forceps, it was with difficulty that the plate could be dislodged. By using a suction tube which could be insinuated between the dental plate and left lateral wall simultaneously, with traction of the plate to the right and movements backwards and forwards, I was able to dislodge the tooth which was lying anteriorly and was acting as a hook. The plate came out after some few moments of jig-sawing. The larynx was further exposed with the Lynch bivalve speculum; the interior of the larynx was then well painted with tincture of benzoin compound because of its antiseptic properties. Time consumed, 12 minutes.

The patient was dismissed from the hospital in three days and is to remain under the care of his local physician with instructions to decannulate the tracheotomy tube after the method first described by Chevalier Jackson.

As you will notice from the X-ray showing removal, there is some evidence of a disturbance of the laryngeal box which may have been caused by the presence of the foreign body in the larynx, but there is also the possibility that a constitutional condition may be the responsible factor.

In my opinion it appears that in similar cases of foreign bodies in the larynx, the suspension apparatus gives considerable advantage in the removal of intruders of large proportion, as it permits one to have the assistance of two free hands and the wide exposure of the throat; it also permits assistance, which can be given by either one or two other physicians as the occasion demands.

I am certain that the suspension method in this particular case obviated the necessity of removing this foreign body by laryngofissure.

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SYMPOSIUM:
THROMBOPHLEBITIS OF JUGULAR BULB.

I.—THROMBOPHLEBITIS OF THE JUGULAR BULB AND LATERAL SINUS OF NONOTITIC ORIGIN. IMPORTANT EAR FINDINGS. REPORT OF THREE CASES.*

DR. SAMUEL ROSEN, New York.

The three cases about to be described are those of jugular and lateral sinus thrombosis of nonotitic origin. The first case is one in which the thrombosis was a retrograde one, extending from a thrombosed lateral sinus of otitic origin to the lateral sinus of the healthy side. The second and third cases are those of retrograde thrombosis of the lateral sinus extending from a thrombophlebitis of the internal jugular vein of nonotitic origin. In the first case a peculiar appearance of the drum was observed on the noninfected side and in the second and third cases a similar peculiar appearance of the drum was observed on the same side as the thrombosis. This appearance of the drum was first noted by Dr. S. Karelitz. Its significance and cause will be discussed by Dr. Maybaum.

Case 1: A child, age 7½ years, had a left myringotomy performed on May 29, 1932, which was followed by a profuse discharge. Four days later there was left hemicrania, pain in the left eye, slight blurring of the fundi and postauricular edema. On June 2, therefore, a simple mastoidectomy was performed. The mastoid was the site of extensive bone necrosis with a large amount of free pus and a perisinus abscess. Mastoid culture taken at operation was pneumococcus Type III. Large granulomata were adherent to the sinus wall. Forty-eight hours after operation the temperature rose to 104.4°. Blood culture done at this time was sterile. For the ensuing two weeks the patient had low-grade fever, some headache and occasional pain in the left eye. On June 18 there was a sudden rise in temperature associated with chill and positive blood culture, indicating the necessity for a sinus and jugular operation. The jugular vein was therefore tied and an occluding thrombus was found in the bulb and lateral sinus. At the torcular free bleeding was obtained. On July 3, the thirty-sixth day of her illness, the child had a convulsion which involved the right side of the body. On July 14 an exploratory craniotomy for an abscess of the left temporal lobe was negative. On

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July 23, about five weeks after the sinus and jugular operation on the left side, this peculiar appearance of the drum on the right side was noted, associated with some swelling in the glands of the neck on this side. On July 27 the left side of the brain was again explored because of bilateral temporal hemianopsia. At this exploration a large abscess of the brain was found. On Aug. 13 the child developed purulent meningitis and died two days later. Postmortem examination revealed that the thrombus of the left lateral sinus had propagated and involved the entire right lateral, petrosal sinuses and jugular bulb.

Case 2: This is a child, age 5 years, who on Dec. 30, 1933, had a right parapharyngeal abscess incised. There was cervical adenitis and fever up to 103°. Forty hours after incising the abscess the child had a severe spontaneous hemorrhage from the abscess cavity. It was necessary to pack the cavity to control the bleeding. On Jan. 2, 1934, chemosis of the right conjunctiva appeared. There was at this time a pinkish discoloration of the right ear drum. The conjunctival chemosis increased and pus appeared in the anterior chamber, necessitating evisceration of the right eye on Jan. 8. After operation the child seemed to be doing quite well except that the congestion in the tympanum increased. This finding will be described by Dr. Maybaum. On Jan. 14 a myringotomy was done, but no pus was obtained. On Jan 16. there was a chill, rise in temperature to 105° and positive blood culture for hemolytic streptococcus. There were no signs of mastoiditis. It was decided, however, to explore the right lateral sinus. Exploration of the mastoid revealed no disease. The jugular vein was found to be completely thrombosed, the thrombus extending upward to behind the knee of the sinus. This child made an uneventful recovery.

Case 3: We are indebted to Dr. Maybaum, Dr. Winkelstein and Dr. Neuhof for the privilege of presenting this case of a boy, age 12 years, who, after a throat infection, developed a swelling of the left side of his neck which was incised Sept. 6, 1935. A deep abscess was found, but the septic temperature persisted and the blood culture was positive for streptococcus hemolyticus. On Sept. 12, because of the persistent sepsis, the neck wound was reopened and an internal jugular phlebitis was found. The vein was tied off, but there was no bleeding from the bulbar end. The symptoms and bacteremia persisted. At this time it was suggested that the ear on the same side be examined because it might show the evidence of a spreading thrombus into the bulb and lateral sinus. The findings in the drum were not unlike those of the other two cases, but nothing further was done surgically because the bacteremia disappeared and the temperature came down to normal and remained so for six days. On Sept. 27 the temperature rose suddenly to 104° and hemolytic streptococcus was again recovered from the blood. At the operation on Sept. 18 the left sigmoidal sinus was exposed. The mastoid was normal. There was an occluding thrombus in the bulb which filled the lateral sinus to within 2 cm. of the torcular where free bleeding was encountered. There was no bleeding from the petrosal sinus. The patient did well for a time but finally succumbed to a purulent meningitis.

Mt. Sinai Hospital.

SYMPOSIUM:
THROMBOPHLEBITIS OF JUGULAR BULB.

II.—THROMBOPHLEBITIS OF THE JUGULAR BULB AND LATERAL SINUS OF NONOTITIC ORIGIN. IMPORTANT EAR FINDINGS. ORIGIN AND EXTENSION FROM THROAT INFECTIONS.*

DR. IRVING B. GOLDMAN, New York.

Retrograde jugular phlebitis derived from the pharyngeal region occurs more frequently than is suggested by case reports. In reviewing the literature, one is impressed by the number of postmortems which show an unsuspected retrograde thrombosis of the internal jugular vein, and the venous sinuses which communicate with it directly or indirectly. Thrombosis of the jugular vein is now well known and surgeons are on the watch for it; the mortality, however, is still high. Any additional aid in the recognition of retrograde extension is significant and important.

Mosher recognized the importance of angina and infection of the jugular vein in 1920 and subsequently made valuable anatomical and surgical contributions to this subject. He realized the importance of the pharyngomaxillary fossa and pointed out that in children this fossa may pick up infections and serve as a route for primary bulb infections. He also suggested that since primary thrombosis of the jugular vein from infections of the tonsil is a recognized fact, there is no reason why primary jugular bulb thrombosis cannot result from the same cause. Today we suspect that there are cases of primary jugular bulb thrombosis which were presumably of otitic origin, but which have been due to a retrograde phlebitis of pharyngeal origin with extension to the bulb. The middle ear changes in these instances are not the cause, but rather the effect of the phlebitis.

The clinical manifestations of jugular phlebitis are not always typical. The primary source in postanginal sepsis

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may be an insignificant sore throat or an abscess in the vicinity of the tonsil. A latent period may follow a tonsillar infection, during which the sepsis develops and the infective primary portal of entry completely resolves. This interval of time between the activity of the primary infection and the appearance of sepsis may vary from a few days to two weeks, but may sometimes be delayed as long as four weeks. There may or may not be a tender gland at the angle of the jaw. The jugular vein may or may not be tender. The first evidence of sepsis may be a metastatic lesion. In one of our cases, the metastasis to the eye was the first and earliest signs of phlebitis. Bone and pulmonary metastases are frequently encountered. Multiple lung abscesses secondary to aspiration of pus from a peritonsillar abscess which has ruptured spontaneously or suppurative pulmonary infarction which has resulted from phlebitis, may occur. Differential diagnosis between phlebitis and pulmonary suppuration may cause sufficient delay so that the patient may die before surgery is attempted. The following case demonstrates this occurrence: A female, age 21 years, had a peritonsillar abscess which ruptured spontaneously four days before admission to the hospital. The soreness of the throat disappeared. She was admitted to the hospital because of chills and fever. The peritonsillar region had almost completely cleared up, except for a small pinpoint opening in the left side of the soft palate which led to the peritonsillar cavity. She ran a hectic course. The differential diagnosis lay between multiple lung abscesses secondary to aspiration of pus, or suppurative infarction resulting from a phlebitis. It was not until after death that the blood culture was reported positive; Gram positive cocci in chains grew in anerobic media. At autopsy, the surface of the lungs was covered diffusely by round, yellow, firm, fluctuant abscesses. Anerobic Gram positive cocci were also recovered from the metastatic lung abscesses. Early exploration of the jugular vein should have been done in this case, but the history of aspiration and the pulmonary picture confounded the situation.

A mild sore throat may become the seat of a severe sepsis because of infection of the regional lymph nodes. These drain into the deep cervical nodes which lie on the internal jugular vein and are sometimes adherent to it. Suppuration of these glands, with the formation of a deep abscess, may cause early and extensive thrombosis because of this intimate anatomical

relationship. This was the method of extension in Case 3. The original focus of infection had subsided and the patient seemed to be getting on comparatively well, while a phlebitis had been developing insidiously. A deep cervical abscess is a perijugular abscess, and with the first signs of sepsis the jugular vein must be investigated. Persistence of sepsis may mean an extension either to the subclavian and innominate veins, or retrograde to the bulb and other venous sinuses. At this point the otitic finding of tympanic congestion first described by Karelitz may be an aid in diagnosing the direction of the extension. Prompt and adequate surgical measures must be taken to prevent complications and fatality.

The superficial cervical nodes lie along the external jugular vein and drain into the deep cervical nodes. A phlebitis of the external vein may be caused by suppuration of these nodes. A phlebitis of the internal jugular vein may then be caused by extension of a thrombophlebitis from the external jugular vein. This vascular pathway of infection to the internal jugular vein, that is, from without inwards, is in contradistinction to the more usual vascular route from within, outwards through the pharyngomaxillary fossa.

An important factor in the production of a phlebitis of the internal jugular vein is the presence of an abscess in the vicinity of the tonsil. This may be a peritonsillar abscess, a parapharyngeal abscess, or a phlegmon undergoing suppuration. The spread of infection takes place through the pharyngomaxillary fossa and sepsis takes place because of the contents in the posterior compartment, namely, the carotid sheath. In our experience, jugular sepsis has been associated with abscesses which have had a paramedian or lateral position in the pharynx. A retropharyngeal abscess which is in the midline tends to gravitate downward and extend to the mediastinum. A retropharyngeal phlegmonous process also tends to extend to the mediastinum. A postmortem examination in a child, age 10 years, who came to the hospital moribund, disclosed a thimbleful of grayish-brown pus when the pharynx was separated from the vertebral column. The posterior pharyngeal wall was red, indurated, and very much thickened. This thickening and induration extended down about the esophagus and tracheal tissue to the superior mediastinum. This pathway of the retropharyngeal infection is due to a fascial expansion, the alar fascia. This fascia completes the anterolateral aspect of the retropharyngeal space throughout

the neck and separates the retropharyngeal space from the anterior compartment of the pharyngomaxillary fossa.

Case 2 was associated with a parapharyngeal abscess which was adequately drained. The sudden rise of temperature, chill, and finding of a bacteremia, made operation imperative. Of additional interest was the relatively benign earlier course and general well-being of the child after the ocular metastasis was treated. Again it must be emphasized that if the local findings in cases of pharyngeal infections are not sufficient to account for temperature, sepsis must be suspected.

Pathogenesis: There are three distinct views concerning the route and method of infection of the jugular vein. These are: First, a hematogenous route; second, a lymphatic route; and finally, by continuity through the interstitial spaces by means of a phlegmonous process.

The hematogenous theory was sponsored by Fränkel, who maintained that there is primary thrombosis of the jugular vein which begins by a direct infection of the tonsillar vein. A progression of a thrombotic process produces a jugular thrombophlebitis with a resulting blood stream infection.

Uffenorde believed that a lymphangitis and a lymphadenitis are the primary processes. He maintains that the spread of infection takes place along the lymph channels to the internal jugular vein or other veins outside the region of the tonsillar fossa. Waldapfel is in accord with Uffenorde. He has also demonstrated that multiple suppurating lymph nodes or lymphatic abscesses may rupture into the jugular vein at several places along the course of the vein and thus cause a phlebitis and thrombosis. Claus also believes that most cases of anginal sepsis originate in abscesses which are found in proximity to the tonsil. The collections of pus spread deeper with the connective tissue, attach themselves to large or small veins and produce a phlebitis.

It is quite likely that various forms of pathogenesis frequently overlap each other. We believe that most cases of jugular phlebitis are due to involvement of small venous radicles in an area of cellulitis, with a resulting thrombophlebitic extension to the jugular vein. Periphlebitis may occur in an area of cellulitis either near or distal to the primary focus. A phlegmonous infiltration of the peritonsillar tissue

may spread by direct continuity to the jugular vein, bathe it in pus, and thus cause a phlebitis.

Despite the contention of many European clinics that whenever a peritonsillar abscess is present, or there is evidence of a general infection, a prophylactic tonsillectomy should be done to avoid complications, attention must be directed to the possibility of converting a transitory bacteremia into a frank bacteremia by trauma. Circulating transient bacteria in the blood stream are clinically insignificant. As a result of trauma, however, these bacteria may cause a local phlebitis and thrombosis. The internal jugular vein may become infected by a thrombophlebitic extension and cause sepsis. That a transient bacteremia plus trauma in a sensitive individual may lead to a thrombophlebitis of the internal jugular vein cannot be denied. Two of six cases reported by Dixon and Helwig on internal jugular phlebitis had early incisions of inflamed tonsils or peritonsillar tissue before sepsis occurred.

Finally, a primary focus of infection for internal jugular phlebitis may occur from any lesion of the pharynx. Among the conditions which may cause extension of suppuration to the perijugular region are infections of the Waldeyer ring of lymphoid tissue, infections of the sinuses, teeth, particularly the last lower molars, the jaws, maxillae, and the parotid gland. These include sources of internal jugular phlebitis which we have not experienced, but which have been observed by others. Pharyngitis or tonsillitis are common infections; it is chiefly the latter, because of proximity to the parapharyngeal space, that is most often responsible for internal jugular thrombophlebitis which, in our cases, lead to bulb and lateral sinus thrombophlebitis.

CONCLUSIONS.

1. Retrograde jugular phlebitis of pharyngeal origin is not uncommon and may be associated with tympanic congestion, which may serve as a clue for the direction of further thrombophlebitic extension.

2. Primary jugular bulb thrombosis may be of pharyngeal as well as of otitic origin.

3. **Consideration should be given to the possibility of converting a transient bacteremia which accompanies acute throat infections into a frank bacteremia by trauma.**

4. **The various pathogenic processes in the development of a phlebitis from throat infections overlap each other.**

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121 East 60th Street.

SYMPOSIUM:
THROMBOPHLEBITIS OF JUGULAR BULB.

III.—THROMBOPHLEBITIS OF THE JUGULAR BULB AND LATERAL SINUS OF NONOTITIC ORIGIN. IMPORTANT EAR FINDINGS. RETROGRADE EXTENSION TO THE JUGULAR BULB AND LATERAL SINUS. DIAGNOSIS AND MANAGEMENT.*

DR. J. L. MAYBAUM, New York.

Sinus thrombosis secondary to a mastoiditis or otitis media presents comparatively little difficulty in diagnosis. In the absence of a history of otitic infection, widespread invasion of the cranial sinuses can occur insidiously and offer a serious diagnostic problem. The three cases presented are of this character.

The purpose of this paper is to awaken an interest in the subject of retrograde thrombophlebitis of the cranial sinuses of nonotitic origin, to draw attention to certain ear findings which may be of diagnostic value and briefly to refer to the management of the condition.

Sepsis due to a thrombophlebitis of the internal jugular vein secondary to tonsillar or parapharyngeal infection has been rather frequently reported in foreign and American literature during the past five years. On the other hand, retrograde extension of a thrombophlebitis of nonotitic origin to the jugular bulb and cranial sinuses has received only casual reference in the older literature and but scant attention in more recent years, and then usually with regard to postmortem findings.

Of the cases reported in the literature of retrograde extension of a thrombophlebitis to the cranial sinuses, many give an identical history of a throat sepsis with or without resection of the internal jugular vein, followed by a continuation

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of the sepsis and discovery at postmortem of a widespread involvement of the cranial sinuses. Postmortem findings in cases of this type have been reported by R. T. Binns,[1] A. Hymanson,[2] H. Rubin,[3] W. P. Eagleton,[4] W. Uffenorde,[5] H. Claus,[6] and others.

We are aware of the difficulty at times of diagnosing a sepsis secondary to a throat infection accompanied by thrombosis of the internal jugular vein, particularly so if few or no manifestations can be elicited upon examination of the neck. From experience with the cases reported, our impression is that a thrombophlebitis of the cranial sinuses of nonotitic origin can easily escape recognition and probably occurs more frequently than has hitherto been suspected. A retrograde spread of the phlebitis may take place over a considerable extent, even to the cranial sinuses on the opposite side far in advance of the thrombus formation on the diseased side, without being recognized. During the course of a retrograde thrombophlebitis of the cranial sinuses there may be, for a considerable interval, complete cessation of evidence of a sepsis. Such was our experience in Cases 1 and 3. How are we to diagnose a jugular bulb thrombosis of nonotitic origin sufficiently early so that operative interference may not be futile? The following data should receive careful consideration in arriving at a diagnosis:

1. A history of a tonsillar or parapharyngeal infection with all the accompanying evidence of a generalized invasion.

2. There may or may not be local evidence of extension of the thrombophlebitis to the internal jugular vein. Such evidence is disclosed on exposing the vein.

3. Suspicion is aroused of possible extension of the thrombophlebitis to the jugular bulb if the internal jugular vein is collapsed or if upon its resection there is an absence of bleeding from the upper stump.

4. Characteristic ear findings such as were present in the reported cases.

5. Involvement of the IXth, Xth and XIth cranial nerves as they pass through the posterior lacerated foramen may give evidence of irritation and later paralysis of these nerves as indicated by the presence of hoarseness, cough, pain on swallowing, vagus pulse, etc. Pain in the eye may be due to a phlebitis of the inferior petrosal sinus.

The characteristic ear findings referred to above were first observed and described by Dr. Samuel Karelitz[7] (Cases 1 and 2). These findings were corroborated by Dr. Samuel Rosen and Dr. Irving Goldman. Somewhat similar findings were observed by me in the third case.

There was a painless involvement of the ear, a bluish or dark red engorgement of the drum starting at the periphery of the lower quadrant and gradually involving the entire drum, and in Case 2 the adjacent canal wall in its entire circumference, was similarly involved. Persistent good hearing and absence of evidence of mastoid involvement were noted. The cervical glands at the angle of the jaw may be tender and swollen. This was the ear picture in Cases 1 and 2. It is too early to state that the ear findings are present in every instance of jugular bulb invasion of this type.

The ear findings in Case 3 differed somewhat from those observed in the first two cases. I was called in consultation to see this boy, age 12 years, a few days after Dr. Neuhof had resected the left thrombosed internal jugular vein. There was no bleeding from the upper stump of the vein. The septic temperature remained unaltered after this operation and the blood culture continued positive. At this time it was suggested that the ear be examined for a possible presence of engorgement from a spread of the thrombophlebitis to the jugular bulb. I observed a dusky red left drum showing a level line reaching midway between the lower pole of the drum and the umbo. The line did not change on movement of the head. The landmarks were present. During the following days there was an increase in the area of drum involved so that the dusky red appearance had reached to the level of lower end of the malleus and the drum posteriorly showed a similar appearance. The canal wall did not show the cyanotic appearance described by Dr. Karelitz in Case 2. As in Cases 1 and 2, there was no pain and the patient heard a low whisper.

The absence of bleeding from the upper end of the internal jugular vein and the appearance of the drum noted above strongly suggested an extension of the thrombophlebitis to the jugular bulb. During the next week the clinical course showed a marked improvement — the temperature did not reach higher than 100° and the appearance of the ear gave evidence of improvement after the eighth day. Severe pain

in the eye one week after the initial observation of the ear findings suggested a possible extension from the jugular bulb to the inferior petrosal sinus. At this time there was definite tenderness on pressure over the posterosuperior cervical triangle. The following day there was a rise of temperature to 104°, preceded by a chill, and the blood culture was positive. The operative findings disclosed a partly obliterative thrombophlebitis extending from the jugular bulb to the torcular. A subsequent benign course for about 10 days was followed by evidence of a right cerebral involvement which ran a distinctly intermittent course for two weeks. The findings upon exploration by Dr. Ira Cohn were negative, except for edema of the brain. It was suggested that a vascular lesion, such as a widespread thrombophlebitis to the normal side, may have accounted for the clinical picture.

The anatomic explanation of the otitic findings is as follows: In Cases 1 and 3 the internal jugular vein thrombosis resulted from a throat infection that had spread upward against the blood stream to the bulb, thus causing venous congestion in the tympanic veins which empty into the jugular bulb. In Case 1 the thrombophlebitis spread from the left lateral sinus to the right (normal side) occluding the right lateral sinus and jugular bulb, leading to tympanic congestion in the same manner as in Cases 2 and 3. Other anatomic factors may be of importance, such as a high, thin dome of the jugular bulb with or without dehiscences in the floor of the middle ear. Politzer[8] stated that some of the veins of the tympanic cavity empty into those of the external auditory meatus by means of numerous anastomotic branches which perforate the tympanic membrane at its periphery. This may explain the canal wall appearance observed by Dr. Karelitz.

It has been suggested that the condition called primary bulb thrombosis may be, in reality, in some instances identical with that discussed in this paper. This clinical entity was originally described by McKernon[9] in 1905. The cases of primary bulb thrombosis reported in the literature and the 20 cases from the Otological Service of Dr. I. Friesner,[10] reported before this section, differ widely from the condition presented this evening. Of the 20 cases reported by us, there was a history of pain in the ear in all and with one exception the ears were discharging. In other words, we were dealing with an acute or a subacute purulent otitis media. In the cases of

nonotitic jugular bulb thrombosis, pain in the ear was entirely absent and the appearance of the ear drums suggested a noninflammatory, congestive middle ear condition. The internal jugular vein in all of our cases was ligated. For the distance exposed, the veins did not give evidence grossly of a phlebitis.

Management: We have not had sufficient experience to definitely formulate the various indications for operative intervention in jugular bulb thrombosis of nonotitic origin. We are aware of the fact that in a majority of instances of bulb thrombosis secondary to a mastoid infection, operation upon the bulb is not necessary. Our impression is that a bulb-thrombosis of nonotitic origin shows a tendency to an insidious and widespread extension to the cranial sinuses and therefore demands prompt surgical measures as soon as the diagnosis is made.

In the management of this condition two types of cases must be considered. First, those in which the internal jugular vein has been resected, followed by a continuation of sepsis and evidence of extension of the phlebitis to the bulb. Such was our experience in Case 3. In a case of this type there is an imperative indication for obliteration of the lateral sinus. Then there are instances in which the internal jugular vein has not been operated on, evidence of local neck involvement may not be present; there may be suggestive signs of jugular bulb involvement. The jugular vein should be exposed and resected if diseased. In this, as in the former type of case, the question as to whether or not bleeding is obtained from the upper stump must be determined at the time of resection of the vein. If the stump of vein is not patent, it should be sewed to the soft parts and the obliteration of the lateral sinus performed as previously recommended.

Operations upon the jugular bulb such as are described in textbooks on otology are not necessary. A complete sinus operation followed to the bulb is sufficient for all purposes. The bulb is curetted, a catheter placed in it and daily irrigations are carried out.

CONCLUSIONS.

Extension of a retrograde thrombophlebitis from the internal jugular vein to the jugular bulb and cranial sinuses can

easily be overlooked in a sepsis of tonsillar or parapharyngeal origin.

Various suggestive facts of the presence of a jugular bulb thrombosis and a possible diagnostic otitic finding are considered.

The ears should be routinely examined in every case of sepsis of throat origin. The ear findings above described in the presence of such a history of sepsis should arouse supicion of the presence of a jugular bulb thrombosis.

A sepsis of throat origin in the absence of bleeding from the upper stump of the resected internal jugular vein, together with characteristic ear findings, is an imperative indication for obliteration of the lateral sinus and drainage of the jugular bulb.

Because of a tendency to an insidious retrograde spread of this form of thrombophlebitis, obliteration of the lateral sinus should be done early and continued toward the torcular for a considerable distance well beyond any evidence of disease.

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ACOUSTIC TUMORS WITHIN THE INTERNAL AUDITORY MEATUS.*†‡§

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This communication deals with the common tumors growing from the VIIIth nerve. It is a well known fact that the average patient with such a tumor makes his first medical visit to the otologist. The most common early complaint is tinnitus, or the patient may complain of attacks of dizziness or of progressive deafness. On the whole, the symptoms vary very little from those often found with toxic disturbances of the VIIIth nerve or even from the deafness and vertigo which are occasionally produced by Eustachian tube catarrh. As a result, some patients with acoustic tumor have a series of inflations, are told to stop drinking and smoking, have their teeth removed or their sinuses irrigated or their septums straightened before they develop the advanced cerebellopontine angle tumor which sends them to the neurologist. According to the neurologist, acoustic tumors produce a characteristic series of signs and symptoms. This is undoubtedly so by the time the patient develops a large tumor, but the cases about to be presented indicate that there are marked variations in the symptoms in the incipient stages. It has been known since the careful work of Henschen[1] that a great many cerebellopontine angle tumors develop within the auditory meatus. Several completely asymptomatic acoustic neurilemomata have been described within the internal auditory meatus, *cf.* Marx[2], Lange[3], Hardy and Crowe[4].

There is a great controversy as to whether the type of tumor under discussion arises from the mesothelial connective tissue or the ectodermal connective tissue of a given nerve, that is, whether they arise from the endoneurium or

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§Two cases of cerebellopontine angle tumor from the Neurological Institute are reported through the courtesy of Dr. Henry A. Riley.

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perineurium or whether they arise from the sheath of Schwann. As a result they are called perineurial fibroblastomata by Mallory[5], Schwannomata by Masson[6] and peripheral gliomata by others. Stout and Carson[7] call these tumors "neurilemomas." The word "neurolemma" has been used to mean both of the sheaths of an axone, but in current American texts it is used to mean the sheath of Schwann alone. Still, the term neurilemoma is the best term available at present for the type of tumor under discussion, if it is borne in mind that this is a new word and a direct derivation from the Greek. It then indicates what no one disputes, *i. e.*, that the growth arises from the nerve sheaths, and it does not commit as to which of the sheaths. The names neuroma and neurinoma (Verocay[8]) are improper for these growths, because they mean "nerve tumor" and "nerve fibre tumor." Acoustic neurilemomata, microscopically, have a characteristic absence of neurites. Typically, they consist of a slow growing connective tissue of two types called A and B tissue, the one compact, the other looser and resembling myxomatous tissue. B tissue is sometimes called microcystic degeneration (Stout[7]). There are few mitotic figures. The cells are of narrow spindle type and they tend to arrange themselves in parallel strands which often curve; here and there they arrange themselves in distinct "palisades." There is often considerable thickening of the sparsely scattered blood vessels. A capsule is usually described, sharply demarcating the tumor from neural tissue, and this is the only part of the tumor where neurites are occasionally found. Some of the smaller growths have been found to occur in or very near to the vestibular ganglion.

So-called meningiomata often have a similar structure, so that in borderline cases it is difficult to tell in which category a given tumor should be placed. The common practice is to call these borderline types meningiomata if they do not seem to arise from a nerve trunk.

Certain fibrous tumors seen in Von Recklinghausen's neurofibromatosis also resemble these tumors (Marx[2]), largely because they are made up of the same cells; however, the growth of these connective tissue cells is more diffuse and tends to run between the nerve fibres, many of which remain within the growth.

So here we have three types of tumor with similar connective cell elements, all given different names, depending on their situation; that is, "meningiomata," if they seem to arise from the meninges; "neurilemomata," if they grow in the nerve and push the fibres aside, or the neurofibromatosis of Von Recklinghausen if they develop between the fibres without pushing them aside. The word neurilemoma is used to describe the acoustic tumors of this communication because it seems to be the most general term available at the present time to express the finer anatomy of the tumors in question; however, the nomenclature of the subdivisions of tumor types changes from time to time as our knowledge increases. As far as acoustic tumors go, there does not seem to me as yet to be enough unanimity of opinion even among expert cytologists for a certainly correct classification according to exact cellular origin.

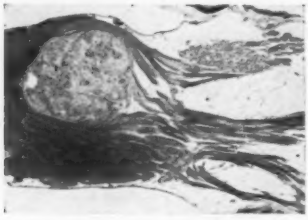

Fig. 1. Case 1 showing small tumor in region of Scarpa's ganglion.

In the last five years, four cases of acoustic neurilemoma have occurred among our pathological sections at the Columbia Presbyterian Medical Center. The first died of peritonitis following appendicitis and had no auditory symptoms except tinnitus. There was no reason to suspect an auditory tumor and no audiogram was made. On microscopic section of the left temporal bone, a tumor measuring 2 mm. on its longest axis and perhaps 1.5 across, was found at the upper edge of the internal auditory meatus and extending into the vestibular portion of the nerve (see Fig. 1). Most of it, however, was above the vestibular and cochlear portions of the VIIIth nerve and lateral to the VIIth nerve. This tumor shows some whorl formation and very little palisading (see Fig. 2). The

cells are a bit large for a typical neurilemoma. It is somewhat like a meningioma, but to me and most of the neuropathologists to whom I have shown it, the tumor resembles the ordinary acoustic tumor type strongly and since it seems to have arisen from a nerve trunk, I have included it in this group.

The second patient was a negress whose terminal illness and cause of death were not very well understood even after autopsy. She had syphilis and some sort of idiosyncrasy to arsphenamine. She gave a history of having suddenly become deaf four years before her demise. There was no history

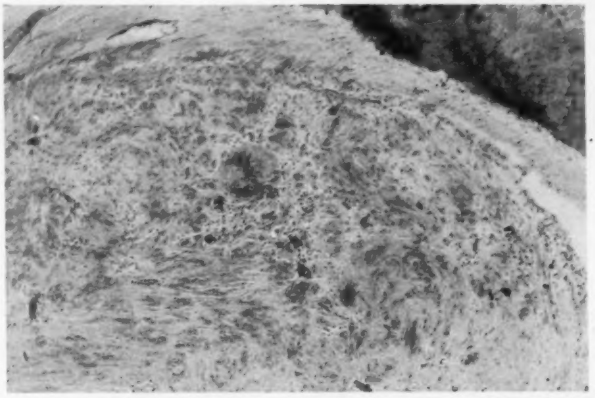

Fig. 2. High power of Case 1 showing whorl formation and palisading; also the fact that part of this tumor growth abuts on the dural lining of the internal auditory meatus.

of vertiginous attacks. Audiometric examination showed complete loss of function in the affected ear. Unfortunately, caloric and rotation tests were not done. On autopsy no lesion was found grossly, but on microscopic examination a small tumor was found, involving the entire base of the modiolus and running about 3 mm. into the internal canal (see Fig. 3). There had been some growth of short fibre web bone in the fundus of the canal and this was attached to the calcified connective tissue of the cribriform plate. There are tumor cells in and about the spicules of bone. These cells are loose and the tissue is more myxomatous than in the peripheral areas.

The main part of the growth, although showing the typical microscopic cell structure of a neurilemoma does not appear rounded and encapsulated as in most reported cases. The cochlea shows complete degeneration of the spiral ganglia and degeneration of the end organs in the utricle saccule and semicircular canals.

The third patient had a more typical history. She had tinnitus and slight deafness for five years. Her chief complaint on admission to the Neurological Institute was attacks of

Fig. 3. Showing small tumor filling up base of modiolus of the cochlea. No growth in facial nerve area or in posterior fossa.

nausea and vomiting over a period of several months. This was followed by attacks of mild dizziness and tingling in the right hand. In the hospital she had signs of intracranial pressure, left lower facial weakness, some nystagmus on looking to the left and deviation of the tongue to the right. She was examined by Dr. Northington who found complete loss of vestibular functions on the affected side. Audiogram showed some loss of hearing on this side, especially in the high notes (see Fig. 4). The audiogram was made without masking the the good ear, but hearing for spoken voice was found to be somewhat more impaired than the audiogram would indicate,

while caloric tests were being made on the good ear. On the other hand, the audiogram would indicate by its form that it was not a "shadow curve" from the good ear. Operation revealed a cerebellopontine angle tumor which was only partially removed. The patient died shortly after the operation and postmortem examination revealed quite a large angle tumor. Microscopic examination of this tumor showed it to have the typical cell structure of a neurilemoma. Serial sections of the temporal bones show that the growth apparently arose near the vestibular lamina cribrosa in the region of the transverse crest (see Fig. 5). The end organs of the vestibule and the ampullae of the semicircular canals are

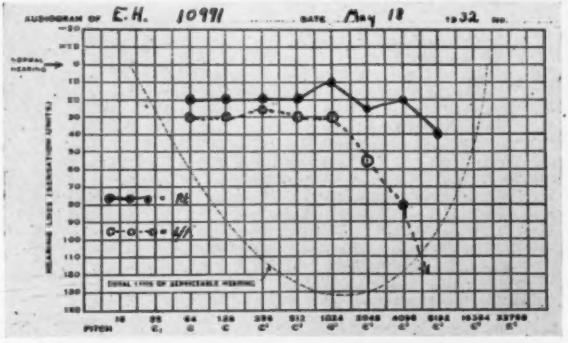

Fig. 4. Audiogram of Case 3 showing moderately good hearing, except in highest notes on the affected left side.

destroyed, as are the end organs in the lower turns of the cochlea. The facial nerve, as it comes to the edge of the transverse crest, seems to be partially invaded by the tumor. Again there is no collagenous fibrous tissue where the tumor comes in contact with nerve tissue, but on the other hand, due to the original density of the growth, there is a sharp delineation from the looser nerve tissue. There was, incidentally, no widening of the internal auditory meatus.

The fourth case was a still more typical acoustic tumor, clinically. On admission to the Neurological Institute the patient had tinnitus, deafness, vertiginous attacks, right lower facial paresis and bilateral involvement of various other

cranial nerves, as well as weakness of the left side of the body. She died postoperatively and was found to have a very large cerebellopontine angle tumor. Microscopic sections show this to have the typical architecture of a neurilemoma. Serial section of the temporal bones show the lower part of the internal auditory meatus to be entirely filled by a fibrous tumor of the same structure as the intracranial mass (see Fig. 6). The internal auditory meatus is somewhat wider than the average, but no bone destruction is apparent in any of the sections and the meatus of the normal side is also larger than average. The spiral and vestibular ganglion cells

Fig. 5. Showing tumor tissue (and postmortem blood) within the internal auditory meatus.

are absent. The end organs in the cochlea and in the vestibular apparatus are entirely destroyed. There is some invasion of the upper part of the fundus of the internal meatus. A few nerve fibres of the superior canal remain. The spaces about them are invaded by tumor, which is not sharply demarcated from normal nerve tissue and vestibular ganglion in this region. The facial nerve is partially compressed by the tumor.

Here, then, we have all kinds of variations in the lesions and therefore the symptomatology produced by acoustic neurilemomata. It would seem that there might not always be a "characteristic" syndrome in the early stages of the growth

since the tumors seem to grow in various portions of the nerve. The early symptoms are dependent upon the exact location of the original growth and whether the speed of growth is rapid enough or irritating enough to produce symptoms from the involved portion of the nerve. The later symptoms depend upon how much other structures within the auditory meatus are compressed or invaded by the growth. It can be seen from Case 3 that quite large tumors can develop in the internal auditory meatus and grow into the cerebellopontine angle without marked compression of either the facial

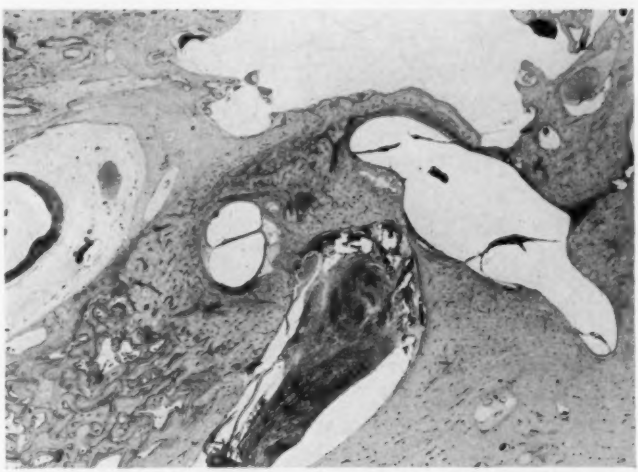

Fig. 6. Showing tumor tissue practically filling the entire internal auditory meatus.

or cochlear portions of the VIIIth nerve. Unfortunately, the involvement of the cochlear portions of the nerve produces the first symptom noticed by the patient. I think it cannot be overemphasized that every case of tinnitus and of nerve deafness is suspect for an acoustic tumor. They should all have audiograms and careful vestibular examinations. Our present methods of examination are still quite gross from a quantitative standpoint, but I venture to predict that within the next few years we will be able to diagnose these tumors while they are still within the internal auditory meatus, and

perhaps an ideal operation may be developed to remove them at this early stage.

The average neurosurgeon is aware that many of these tumors arise within the internal auditory meatus, but very little attempt has been made, to date, to remove the pedicle in the internal auditory meatus during the operation. This is largely due to the technical difficulties of removing the tumor *in toto*, but these pathological studies indicate that the point of origin is from the fundus of the internal auditory meatus and that, therefore, every effort should be made to

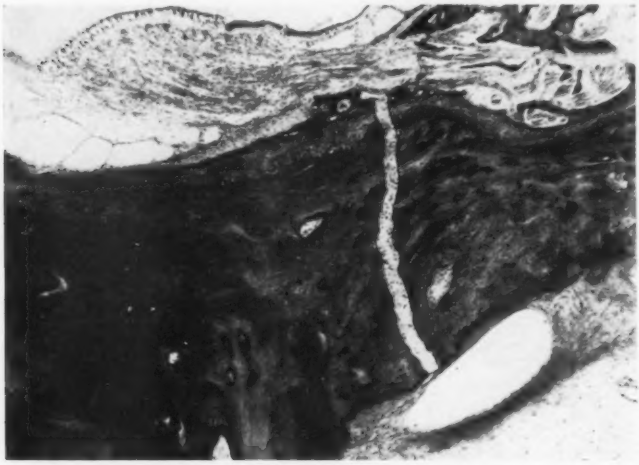

Fig. 7. A symptomatic fracture through the cribriform plate in the region of the macula of the utricle, from a specimen presented to the author for the American Otological Society by O. Mayer, Vienna.

develop a technique which would remove the growth from the lamina cribrosa.

Studies of serial sections of temporal bones in which the growth is found in the region of the porous acousticus, engender a theoretically possible etiology for certain acoustic neurilemomas. In the past 10 years there has occurred in the otological literature (O. Mayer[9], Brunner[10]) a series of reports showing microscopic fractures in the temporal bones of middle-aged and old-aged people, the age group which develops

acoustic tumors. A great many of these microscopic fractures occur in the cribriform plates themselves, and most particularly in the vestibular cribriform plate (see Fig. 7). The reason for this may be the fact that the temporal bone is weakest in this region because the internal and external auditory meati are here on the same line; and, furthermore, the bone of the cribriform plates is of a very brittle type. It is more a calcified connective tissue than a true bone. It is conceivable that the same forces which produce these microscopic fractures may stretch some of the fibres of the VIIIth nerve over the sharp edges of the small holes in the laminae cribrosae or the transverse crest and so break their primitive sheath or their endoneurium. Incidentally, the protective connective tissue coat of the nerve fibres becomes much thinner just as they enter the cribriform plate. Studies of cut and injured nerves show that when the sheath of an axone is traumatized, there is often a tremendous proliferation of the cells of the endoneurium or sheath of Schwann or both. In traumatic injuries of other nerves there is usually regrowth of neurites, but an even larger regrowth of Schwann sheaths and endoneurial elements. Eventually, a technique for differentiation of Schwann cells will be developed, then perhaps the exact origin of the specific cells in nerve tumors will be determined. It is irrelevant for our hypothesis, however, whether it is the endoneurium or the sheath of Schwann which proliferates. It suffices to say that histologically acoustic neurilemomata resemble certain traumatic neuromata very closely (Masson[6]). The only difference is that most acoustic neurilemomata contain very few, if any, neurofibrillar elements. That might be explained by the fact that in experimental cutting of the VIIIth nerve, no regeneration of neurites has ever been known to occur. The neurofibrils, occasionally found in the edge of acoustic neurilemomata, may very well come from partial severance of the facial nerve. This theoretically possible explanation of the cause of acoustic neurilemomata will have to be proven experimentally on animals or perhaps some case of surgical severance of the VIIIth nerve for Meniere's disease will develop a cerebellopontine angle tumor.

Another anatomical fact of possible importance in the etiology of acoustic tumors, especially the type which resembles the so-called meningiomata, is the common occurrence of psammoma bodies and of enlarged arachnoid villi within the

internal auditory meatus. If the theory that meningiomata originate from these structures in the falx and elsewhere is correct (Mallory[5], Cushing[11]), then certain acoustic tumors may originate from these structures within the auditory meatus.

There is still another academic matter of interest to otologists which is brought out in these studies. In spite of the partial or complete degeneration of the VIIIth nerve in these sections, no otosclerotic foci were found in the bones. This would tend to cast some doubt upon the hypothesis of the late A. Gray[12], who felt that otosclerosis is caused by degenerative processes in the VIIIth nerve itself. It suggests that the cases of otosclerosis which he found in conjunction with acoustic tumors were chance findings. I have seen absence of neural elements, such as he describes, in the VIIIth nerve with normal facial nerve in cases of otosclerosis, but only in cases where there had been marked pulling on the nerves during postmortem removal of the brain.

More important than these hypothetical considerations is the report of the cases themselves. They emphasize that acoustic neurilemomata occur at the very bottom of the internal auditory meatus where they can produce degeneration of the cochlear and vestibular end organs without ever getting out of the canal (Case 2), or they may spring from the typical site and produce a large cerebellopontine angle tumor without interfering with facial or very much with cochlear function (Case 3). On the other hand they may produce no symptoms at all (Case 1). Or they may involve most of the cranial nerves and several of the spinal tracts (Case 4). The pressure symptoms, therefore, vary considerably, but it must not be forgotten that the probable primary site is deep inside of the temporal bone.

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CRANIOCERVICAL MOVEMENT AND MUSCLE STRENGTH.*

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In a previous paper the writer indicated the need for the study of the craniocervical structures from the reflexologic standpoint.[1] It was furthermore shown that in ear and mastoid diseases and their intracranial complications, as well as in various neurological conditions, diagnosis is facilitated by a knowledge of craniocervical mechanisms.

Manipulations of the head and neck in mastoid and cervical involvements, in vestibular tests as well as in the diagnosis and treatment of upper respiratory affections, all call for a better understanding not only of the reflex mechanisms but also of movement and muscle strength of the craniocervical neuromuscular apparatus. The hyoid bone and thyroid cartilages contain numerous delicate muscles that form part of this system and are intimately concerned in head movement aside from their more particular association with deglutition and phonation.

Except for the cephalogyric paralysis that occurs as part of the syndromes of Schmidt and Jackson and the occasional references to the nuchal musculature in the elicitation of the tonic neck reflexes, little attention has been paid by the neurologist to the cephalocervical region; therefore, it is advisable to dwell upon the physiology as well as the clinical neurology of this region.

ANATOMY AND PHYSIOLOGY.

The movements of the head consist of flexion, extension, lateral, anterolateral, posterolateral, and rotatory. The anterolateral and posterolateral are partially rotatory since both the anterior and lateral, or the posterior and lateral muscle groups are involved.

Flexion of the head is illustrated in Fig. 1. It is produced by the rectus capitis anterior and the hyoid group.[2] This is a

*From the Neurological Service and the Neurological Clinic of the City Hospital.

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bilateral movement since the above muscles of both sides are utilized when the head is flexed in the normal individual. The innervation of these is from the first to the fourth cervical metameres and by the facial.[3]

Fig. 1. Flexion of the head or the anterior craniocervical movement.
Fig. 2. Extension of the head or the posterior craniocervical movement.

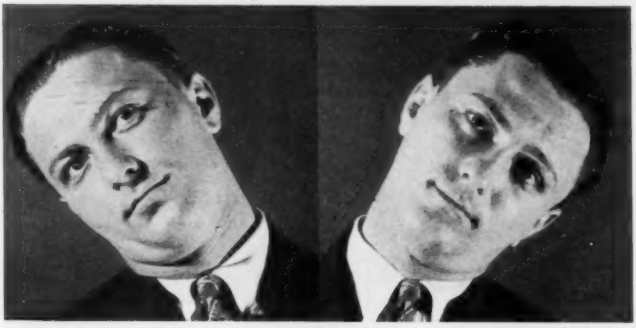

Fig. 3. Right lateral craniocervical movement.
Fig. 4. Left lateral craniocervical movement.

Extension of the head is illustrated in Fig. 2 as the posterior craniocervical movement. It is produced by the splenius capitis, longus capitis, complexus, obliquus inferior, and rectus capitis posterior major and minor muscles. These are innervated largely by cells from the first two segments of the spinal cord;[4] this movement is also a bilateral one since

in the normal individual the muscle groups of both sides of the neck act when the head is extended.

Lateral motion of the head is illustrated in Figs. 3 and 4. It is produced by the splenius capitis, longus capitis, semi-

Fig. 5. Elicitation of anterior craniocervical muscle strength.
Fig. 6. Elicitation of posterior craniocervical muscle strength.

Fig. 7. Elicitation of right lateral craniocervical muscle strength.
Fig. 8. Elicitation of left lateral craniocervical muscle strength.

spinalis capitis, obliquus capitis superior and rectus capitis lateralis muscles. Although some of the muscles in this group also produce extension of the head, the lateral movement is strictly produced by the muscles of the side in which direction the movement is executed.

Rotation of the head is produced by the combination of some of the above in the following group: Splenius capitis, complexus, obliquus capitis superior and inferior, and rectus capitis posterior major and minor. The execution of this movement requires the normal function of all muscle groups

Fig. 9. Elicitation of right anterolateral craniocervical muscle strength.
Fig. 10. Elicitation of left anterolateral craniocervical muscle strength.

Fig. 11. Elicitation of left posterolateral craniocervical muscle strength.

of the craniocervical area. It requires rotation of the head upon the atlas in all directions so that flexion extension lateral and combinations of these are executed. This should be distinguished from the hinge-like action as produced by the sternocleidomastoids alone and where the face moves from

side to side and where the vertical axis through the head and neck is not tilted. The innervation of these is by the cervical segments[2] and by the spinal accessory nerve.[5]

The anterolateral and posterolateral movements which may be noted in the figures of such illustrating muscle strength in these directions are also carried out by the last group and hence have the same innervation. These movements are purely unilateral since the right or left lateral groups of muscles are brought into action in association with the anterior or posterior muscle groups of the same side.

The muscle strength of the particular group of the above is elicitated as anywhere else in the body by the production of opposition. Thus the muscle strength of the anterior craniocervical group is tested by producing opposition to flexion of the head as is noted in Fig. 5. That of the posterior craniocervical group is illustrated in Fig. 6, *a.* and *b.* side view and back view, respectively. The elicitation of the right and left lateral craniocervical muscle strength is illustrated in Figs. 7 and 8 respectively. The method of eliciting the right and left anterolateral muscle strength is illustrated in Figs. 9 and 10. Both the anterior and right and left lateral muscle groups are respectively simultaneously tested. This is likewise true for the posterolateral positions where extension of the head is associated with lateral action of the right side as in Fig. 10, or with posterolateral opposition from the left (see Fig. 11).

The above movements and tests of the muscle strength were carried out in many normal individuals. In general the muscle strength is almost equal in all directions in the average person with the exception of the flexion of the head where the strength seems to be somewhat less than that noted while force was exerted with the head in the other positions.

CASE PRESENTATION.

In the following cases it will be shown how muscle strength and movement in the craniocervical region are affected in various intracranial, spinal and peripheral nerve diseases.

The first case deals with effects of meningeal irritation upon the craniocervical region.

Case 1: S. J., white, male, age 16 years, was sent from the City Hospital Medical Service to the Neurological Clinic.

He complained of a right-sided weakness, numbness and generalized pains. He was treated for a meningococcus meningitis at the hospital. His family history is irrelevant.

The neurological examination showed a slight disturbance in the Romberg and some hesitation in the finger to nose test. His tendon reflexes were somewhat unequal. His right scapular reflexes[6] were greater than the left. The right facial reflex[7] was greater than the left. The left abdominal was greater than the right. Sensation showed no disturbance except for hesitation regarding the comparative intensity of the stimuli. The pupils were sluggish to light. In the cervical and lumbar region there were areas of muscle tenderness and slight sensory changes.

The diagnosis was radiculitis and myositis following meningococcus meningitis.

The craniocervical muscle strength was markedly diminished anteriorly and laterally on the right side, while posteriorly and laterally on the left side it was greater. This is probably the residuum of the inflammatory effect that the meningeal involvement had upon the craniocervical sensory[1] as well as motor components. Many cases of meningitis with the characteristic nuchal rigidity were seen. This rigidity often bore a direct relationship to the intensity of the involvement. The anatomical basis for such a reaction that has merely been used as a "sign" of meningitis has been definitely illustrated.

In the following case it will be shown how a purulent inflammatory process affecting the pyramidal system diminishes the muscle strength of the craniocervical musculature.

Case 2: S. P., white, male, age 16 years, was transferred from another hospital to the neurological wards of the City Hospital with the following history: The patient suffered from a chronic otitis media of the right ear of six years' duration. Six months ago he developed a left hemiplegia and was diagnosed as having a brain abscess for which he was operated. Since the operation he developed some occasional abnormal involuntary movements in the left upper extremity, an aphasia and unprovoked spasmodic emotional upsets, consisting of unprovoked involuntary movements indicating silliness, motor reactions to lugubriousness and crying spells.

Neurological examination disclosed a bedridden patient suffering from a left hemiplegia. Weakness was present in

the left upper and lower extremities in which the tendon reflexes were hyperactive. There was a positive Babinski toe sign and a positive heel sign on the left. Some hypesthesia was also present on the left side, so that tickling the left axillary region failed to elicit the smiling response which was easily elicited by stimulating the right side. A left facial weakness was noted during the involuntary affective motor responses. The right facial tendon reflex reaction was greater than the left, and the winking responses were absent bilaterally. There was moderate disc pallor bilaterally on ophthalmoscopic examination.

The diagnosis was meningoencephalitis and brain abscess complicating an otitis media and mastoiditis.

When the patient resisted the examiners' hand as it was pressed against the left side of his head, there was a weakness as compared with the resistance on the right side. The strength of extensor muscles of the head and particularly those concerned with left lateral motion such as the rectus capitis lateralis splenius capitis, obliquus capitis and sternocleidomastoid, were markedly affected. The left lateral movement of the head was slightly limited.

The following case is illustrative of a great number of cases of hemiplegia or monoplegia that result from cerebral hemorrhage, thrombosis or embolism.

Case 3: C. H., colored, female, age 66 years, was seen at the City Hospital Neurological Ward. The patient has been in the hospital for seven years and has been ill for 10 years, starting with a sudden onset of paralysis of the left upper and lower extremities. Her past history showed that 16 years before admission she had rheumatism. She is married and has three children.

Neurological examination showed a woman with hemiplegic attitude who was emotionally depressed. Touch and vibration were diminished on the left. Muscle power was greater on the right than on the left. The pupils were contracted bilaterally. A paralysis of the left lower face is present. Her tongue was deviated to the left. There was a hypesthesia and hypalgesia of the left side of the face. While at the hospital she had an acute recurrent otitis media on the left. Her

blood Wassermann and urine were negative. Her feces culture was negative for typhoid.

The diagnosis was thrombosis in the region of the right internal capsule and hypertension.

The craniocervical muscle strength was definitely diminished on the left side. Motion was considerably limited on that side as compared with that of the other side. When the patient resisted the examiner's hand anteriorly the tip of the chin rotated to the left, thus indicating a weakness of the left rectus capitis anterior major and minor muscles as well as of the left hyoid group. There was also a diminution of muscle strength in the left anterolateral and posterolateral directions. Here it should be particularly noted that the left lower facial muscles were paralyzed and so was the left side of the tongue.

The craniocervical movement and muscle strength were mostly affected in individuals who suffered from a paralysis of the facial and hypoglossal muscles of the same side. The anatomical relation of these structures explains the clinical neurological findings. Over 50 cases of hemiplegia due to capsular or other lesions, *i. e.*, cortical, or subcortical, were observed. In every case of hemiplegia or monoplegia, particularly if the upper extremity was involved, there was a corresponding involvement of the movement and muscle strength of the craniocervical muscles. The intensity of the weakness or limitation of activity of the muscles of the craniocervical group fell in line with the degree of involvement of the other muscles on the same side. The writer saw one case of cerebral hemorrhage in an adult in whom only the right facial, hypoglossal muscles, and the right craniocervical structures showed evidences of paralysis.

In the following case it is shown how muscle weakness resulting from changes in tonus brought about by involvement of the cerebellar pathways manifests itself in the course of the function of the craniocervical muscle group.

Case 4: J. C., adult, male, age 55 years, was admitted to the Neurological Service of the City Hospital complaining of a weakness of the left upper extremity. Two years ago he had an attack of left hemiparesis. This was not associated

with coma or convulsions; it was sudden in onset. The family history was irrelevant.

Examination showed a coarse tremor of the left upper extremity. Sensation was intact. There was an ataxia with the left finger to nose test and heel to knee test. There was an adiadokocinesis on the left side. The right pupil showed no reaction to light and slight change in reaction to accommodation. The right IIIrd nerve was involved. There was a left supranuclear facial paralysis and the left IXth nerve was involved since the uvula deviated to the right. His NPN was 30 and sugar 190 mgm. per 100 cc. The blood pressure was 120/80.

The diagnosis was Nothnagel syndrome due to a cerebral thrombosis.

There was a marked weakness with the right lateral craniocervical muscle strength test. The left anterolateral was also weak but the right anterolateral reaction was normal.

A case showing multiple tegmental lesions of the pons and medulla due to a thrombosis of both posterior inferior cerebellar arteries presented similar findings to those noted in the above case. The craniocervical muscle strength and movement was, however, bilaterally affected. In this connection it should be noted that many syndromes dealing with the tegmental involvement of the midbrain, pons and medulla will present similar reactions to those noted in the case.

Case 5: J. R., adult, male, age 34 years, came to the clinic complaining of a tremor of the right hand and leg which began in 1922. He had been very sleepy since then. The past personal and family histories were irrelevant.

The neurological examination disclosed a patient showing a slight involvement of the facial muscles, some limitation of automatic associated movement and a tremor of the right hand and leg.

The diagnosis was encephalitis with an early Parkinsonian syndrome.

Craniocervical movement was limited on the back and sides. He complained of stiffness on attempting to bend his head in these directions. There was a greater resistance in the right lateral and antero and posterolateral aspects than on

the left side, when the examiner's hand opposed his head in these directions. Many cases of lethargic encephalitis were studied. The characteristic feature in all of the cases was that individual muscle groups, or combinations of these, presented weakness and limitation of movement with no regard to homolaterality. Sometimes only a few muscles were involved, limiting the head movements in a particular direction. At other times, depending upon the extent of the encephalitic process, the head was immobile in all directions. This was characteristic of all types of encephalitis whether they were due to lues, other types of infection or trauma.

Case 6: J. W., adult, male, age 44 years, came in complaining of gait difficulty, numbness of the feet, tremor and lightness in the head, urinary control impairment and smell disturbance, all of which were of seven years' duration. He had visual difficulty for two years and intermittent sexual weakness. His past personal and family histories were negative.

The neurological examination disclosed a gait difficulty. The right upper extremity was ataxic and both lower extremities showed a spastic paralysis. There was a tremor of the head. The radial and triceps reflexes were unequal and the biceps, KJ and AJ, were exaggerated. He had a right-sided Babinski, Gordon and Chaddock. There was also a positive Rossolimo, heel tap[8] and a left-sided shoulder tap.[9] The external oblique reflex was absent on the left,[10 11] and the abdominals and cremasterics were likewise absent. The sensory examination was normal except for an absence of vibratory sense in the anterior of the right leg. The position sense of the left toe was slightly impaired. The cranial nerves showed a left facial weakness with absent left facial reflexes;[7] the right were present. An optic atrophy and nystagmus were also present.

The diagnosis was multiple sclerosis.

The craniocervical muscle strength was normal, but when the tip of the jaw on the left side was tapped, there were a number of slight movements, resembling those of a swinging door, showing instability of the head on the atlas. This did not occur when the right side was tapped. These movements were rapid but not so rapid as those seen in nystagmus. There were between eight and 10 movements in all, the chin

stopping at seven o'clock and going over to about four o'clock, if we could imagine the lower jaw bone to take the place of the hand on a horizontally placed timepiece.

A case of syringomyelia in a woman was seen. She presented definite pain and temperature dissociation and an atrophy of the muscles of the left side of her neck. The craniocervical examination revealed the following: There was very little reduction in muscle strength except in the nuchal musculature on the left side posteriorly. Although there was no disturbance in head movements, there was definite weakness when the patient was asked to resist the pressure on the hand which was placed in the left posterolateral direction.

Case 7: L. V., adult, male, age about 45 years, was transferred from another hospital to City Hospital with a diagnosis of encephalitic chorea. His past history was as follows: Smallpox in 1914; mumps in 1918; malaria in 1920. He received salvarsan injections. The family history was irrelevant.

The neurological examination showed athetoid movements over the entire body and a positive Romberg. He was unable to lift his feet off the ground while walking. The deep reflexes were absent except for his right biceps. The abdominals and corneals were active and equal. There was no Babinski and there was a slight ataxia in the finger to nose and heel to knee tests. Muscle power in general was weak and his grip likewise was weak. Sensory examination disclosed a widespread disturbance in pin prick, touch, vibration, hot and cold from feet to neck, front and back, irregularly distributed. The laboratory findings were as follows: Blood Wassermann, spinal Wassermann were negative; sugar 91 mgm. and NPN was 25 mgm. per 100 cc.

The diagnosis was peripheral neuritis due to arsenical medication.

The craniocervical examination showed the following: His head was unsteady on his shoulders, and when asked to extend, flex or move it from one side to the other, he did so only partially. When the muscle strength was tested in these directions there was only a slight resistance on flexion and none on the sides or back.

In the above case presentations the effects of cerebral, mesencephalic, pontine, bulbar, spinal and peripheral lesions upon the craniocervical structures were outlined. It was also shown how diffuse lesions of encephalitis vary in their effect upon craniocervical movement and muscle strength depending upon the extent and location of the intracranial inflammatory process. Over 200 cases belonging to the above groups were observed. In addition to the above, the writer examined cases of torticollis, cervical myositis secondary to upper respiratory infection and some cases of traumatic and infectious myositis following cervical abscesses and gland operations. Osteoarthritis of the cervical vertebrae with their associated deformities and radicular syndromes were differentiated by their local signs and pain on pressure from the reactions obtained in cases of central nervous system involvement.

CONCLUSIONS.

1. Due to the frequent recourse to manipulations of the head and neck by the otolaryngologist and neurosurgeon, a proper understanding of the neuromuscular mechanism of the craniocervical apparatus seems to be indicated in both fields.

2. Head movements depend upon the action of muscle groups whose innervation is supplied by cranial and spinal nerves.

3. The muscle strength, as well as motor ability of these muscle groups, is brought into play when the head is flexed, extended, bent to either side, rotated or when flexion and extension are independently combined with lateral motion.

4. Flexion and extension of the head are executed by the action of the bilateral muscle groups carrying this function.

5. Lateral motion of the head is executed by the unilateral group of muscles on the side to which the head is bent. The anterolateral and posterolateral craniocervical movements are likewise unilateral in type in contrast to the anterior and posterior or flexion and extension of the head in which the muscle groups of both the right and the left side act in unison.

6. A better understanding of the types of nuchal rigidity of meningitis and meningoencephalitis, as well as that result-

ing from the tension of increased intracranial pressure upon the meninges, is gained when the mechanism of the craniocervical apparatus is analyzed.

7. Cases of meningitis, brain abscess as well as vascular, and luetic lesions of the cerebral hemispheres were presented, and the effects upon the craniocervical muscle strength and movement were analyzed.

8. A case of hemiplegia due to a cerebral thrombosis was presented and the abnormal craniocervical movements as well as the diminution in the craniocervical muscle strength on the same side were shown.

9. The craniocervical movements and muscle strength are particularly affected in lesions that involve the supranuclear motor representations of the lower cranial nerves.

10. When the head of the patient opposes the examiner's hand in the anterior aspect, the patient's chin rotates towards the hemiplegic side and contralateral to the lesion.

11. Head movements that occur in irritative and destructive lesions of the brain should be explained on the basis of the function of the craniocervical system.

12. A case with a Nothnagel syndrome was presented to show the effects of an extrapyramidal lesion upon the function of the craniocervical structures.

13. The disturbances in movement and muscle strength of the craniocervical apparatus were furthermore illustrated in multiple vascular lesions of the pons and medulla and compared with the reactions obtained in encephalitic involvements of these regions.

14. Different reactions were obtained in cervical spinal diseases depending upon the columns affected. Thus in the anterior horn pathology, associated with a case of syringomyelia, a different response was elicited from that noted in a case of multiple sclerosis.

15. In a case of multiple sclerosis a clockwise and counterclockwise unilateral head clonus, the first one to be presented in the literature, was observed.

16. Lesions of the peripheral cervical nerves produce a much more circumferential and diffuse muscle weakness than that

resulting from the localized intracranial unilateral lesions. They also differ distinctly from the effects upon selective nuchal muscle groups as encountered in encephalitis.

17. Cases of torticollis, myositis, or cervical osteoarthritis can be differentiated from the craniocervical disturbances of the preceding groups by palpation, local tenderness, etc.

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1114 Ward Avenue.

MILK INJECTION FOR PHARYNGEAL AND LARYNGEAL INFECTIONS.*

DR. WARD C. DENISON, New York.

Milk injections as a treatment for ocular and orbital infections have been used for several years with more or less varying results.

Milk has been classified as an animal protein included with egg albumen and horse serum. Considerable literature has been written about proteins employed, such as the animal, vegetable and bacterial forms.

August Lumiere and others in the early work on protein therapy spoke of the "colloid chemistry and the colloidal state" of given substances, but later spoke of "the mechanism of the mysterious colloids that holds in suspense the future progress of biology."

As a field of research, foreign protein therapy is no longer opposed by the standard of immunity.

Ehrlich's side chain theory may best explain this specific mode of action of various antibodies. Now there is a growing tendency to explain many of these reactions as physicochemical and colloidal bases. Antigens are substances that cause antibodies in the body fluids and without exception are colloids and usually proteins in nature. Furthermore, antibodies are colloidal in their chemical characteristics. While they may or may not be solutions of colloids, they are in the final analysis products of cellular activity and, therefore, derived from colloids.

This study of protein therapy, then, embraces the question of anaphylaxis, cellular sensitization and desensitization, immunity, as well as the physicococchemical relation to infections.

A few years ago I saw a patient with a severe pharyngeal and laryngeal infection with swelling and edema. In despair I gave an injection of milk and in a few hours, much to my surprise, her general condition had greatly improved.

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Since that time I have had occasion to use this method of treatment on a number of patients with pharyngeal and laryngeal infections with very satisfactory results.

Among these cases the various types of organisms found have been in the following order: *1.* Streptococcus hemolyticus and nonhemolyticus; *2.* micrococcus catarrhalis; *3.* staphlococcus aureus and albus; *4.* pneumococcus 1, 2, 3, and heterogenous types; *5.* leptothrix, and *6.* streptothrix occasionally. Pneumococcus and streptococcus produce the acute hypermenia and edema, with the latter being the most common offender.

Technique: The technique of administering milk injections is one of simplicity. Whole cow's milk is boiled for three minutes and then 10 cc. is withdrawn into a Luer syringe. A needle with a fairly good-sized lumen is used and the milk is injected intramuscularly into the buttocks. With children under 8 years of age 5 cc. is injected instead of 10 cc. which is the adult dosage.

We formerly gave a dermal test, injecting one drop of milk subcutaneously and then waiting 30 minutes to observe whether the patient was sensitized to the protein. This has been discontinued as an unnecessary procedure.

So far I have had no cases with serum sickness and no ill effects from its administration. Occasionally we have had an increase in temperature, which has only lasted for a few hours.

If the patient has not shown a definite improvement the injections are repeated in 24 hours. In some cases I have waited 48 hours before repeating the treatment. Three doses have been the most that I have been required to give.

The advantages of this form of treatment are as follows: *1.* Milk is always accessible and found in practically every home. *2.* It can be given immediately without delay while laboratory tests are being made as to the type of organism responsible for the infection. *3.* It can be given in conjunction with other medications without contraindication.

I would like to report a case that came under my care some time ago.

Mrs. W. L., female, age 46 years; difficulty in swallowing, acute soreness of the throat, cough, general malaise and a temperature of 102°.

Past History: Nonessential.

Physical Examination: Heart and lungs normal; hypermenia and edema of larynx, pharynx, nasopharynx; hypermenia of nasal mucous membrane; whitish patches over both tonsillar fossae.

Blood Count: R.B.C., 4,800,000; H.G.B., 98 per cent; W.B.C., 21,000; polys., 78 per cent; L.L., 14 per cent; S.L., 6 per cent; eosinophiles, 1 per cent. Throat culture and smear showed streptococcus hemolyticus predominating.

The treatment given at that time consisted of half-hourly saline irrigations and local applications of neosilvol.

The following day the edema and hypermenia had progressed and the whitish patches with a membrane were enlarged. The larynx and nasopharynx was very edematous and the patient complained of shortness of breath and difficulty of swallowing. The temperature was 104°. Due to the fact that the breathing was so labored and the edema had progressed, a tracheotomy was seriously considered.

Ten cc. of whole milk, boiled three minutes, was then injected intramuscularly in the buttocks. The following day the acute hypermenia and edema had subsided considerably and the membrane had practically disappeared. The patient seemed greatly relieved and was able to swallow and breathe without difficulty. The temperature had dropped to 100°. In general, the patient was very much improved. Forty-eight hours later, although possibly not necessary, another milk injection of 10 cc. was given. The following day the hypermenia and edema had practically subsided and no patches or membrane was seen. The patient was comfortable and the temperature had dropped to normal.

I feel very confident that if the milk injections had not been given, a tracheotomy would probably have been necessary, with possibly fatal results. In conclusion, I hope that in the future milk injections will be given in cases of laryngeal and pharyngeal infections, with possibly the very good results which these cases have received.

REFERENCE.

BEN WILT KEY: Protein Therapy in Practice.

20 East 53rd Street.

NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of May 20, 1936.

(Continued from July issue.)

Twenty Years Experience with Iodine Powder (Sulzberger) in the Conservative Treatment of Aural and Nasal Suppuration. Dr. M. D. Lederman.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HENRY M. SCHEER: I want to take just a few minutes to tell of my experience with the use of iodine powder during the past 15 or 20 years in these chronic discharging ears. I have not had any experience with the powder in the nose work. I make it a practice, unless a patient comes in with a very acute, urgent condition that requires immediate operation, where the history is of a chronic purulent otitis media, to first X-ray the patient to decide the extent, if any, of any pathological involvement in the mastoid process. That gives us a picture of the mastoid bone at the time the case is taken on. Then, if nothing urgent is present in the way of pathology, I suggest to the patient to submit to conservative treatment. Conservative treatment to me means the application of dry cleansing of that ear by one of a dozen methods, namely, the use of ether, or alcohol, or any other antiseptic solution, into the middle ear if one can reach the middle ear through the membrane perforation, and into the mastoid cavity if mastoidectomy has been done previously. I follow that cleansing with drying and then in turn use the iodine powder. That treatment applied occasionally is not sufficient. That treatment applied once in three or five or seven or 10 days is not sufficient. At the beginning of the treatment I remove any lesions such as a polyp or granuloma arising in such an area where its removal is not a serious matter. Occasionally in removing a polyp we may set up an acute meningitis, which may occur when removing a polyp from the middle ear which arises from the region of dehiscence of the tegmen tympani. If it can be removed safely, I remove it and follow with the cleansing and drying treatment.

As I said before, following this treatment at long intervals is not sufficient. I use it daily until the discharge is reduced to a minimum and then every 48 hours until the ear is dry. I have made it a rule that if the ear is wet for anywhere from three or four months under that treatment, I do not think it is going to get dry in any other man's hands with conservative measures. I also tell the patient that unless acute symptoms arise with reference to intracranial complications, no surgery should be done. If at the end of the treatment, an arbitrary period of three to four months, the ear is still wet, then I advise the patient one of two things: If he wants to go along with the discharging ears, to stay under observation fortnightly or at longer intervals, provided that he is not becoming more and more deaf and provided there are no intracranial complications; if he does not want to go along with the discharging ears indefinitely, then he should undergo surgery. I do not have to tell you men how urgent it is to teach the general practitioner not to tell his patient with the discharging ear "to go away" and "it will dry up in a little while." It is surprising how many men will discharge such patients and tell them not to worry about it, that the ear will dry up, and how a few years later sinus thrombosis or other serious intracranial complications may occur. It is, therefore, up to us to teach the general practitioner how to treat discharging ears following otitis media acuta.

I can say that this treatment, if applied properly, will give you a dry ear in anywhere from 65 to 70 per cent of cases.

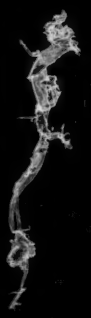
DR. M. J. MANDELBAUM: In March, 1929, I reported in THE LARYNGOSCOPE a series of cases in which I endeavored to find out the diagnostic value of lipiodol introduced through perforated eardrums, and as a result I was very much surprised at the improvement which took place in a considerable number of these cases which were chronic otitis media associated with chronic mastoiditis. A series of seven cases had undergone radical mastoidectomies without complete relief of the odorous discharge. Four of these cases were cholesteatomatous, and three had already been booked by competent otologists for radical operations. These patients were not cured but they were markedly improved by the iodized oil.

Many years ago I heard Dr. Lederman read his paper and did not pay much attention to it. In looking over the literature at the time I read my paper, among the references to the use of iodine for middle ear suppuration, I reminded myself of his work. I must say I am more than impressed with the value of the method, and that is from an absolutely impartial standpoint. I have collected from my own cases and other members of the staff a series of 114 cases treated in the last seven years with the iodine powder (Sulzberger). We had a very good good check-up and have been able to follow up 98 cases. My figures showed that 90 per cent of these cases were cured. Out of the 98 cases, seven had had radical mastoidectomies and six of those seven were absolutely relieved. When I say they were cured, I mean they had been free from the discharge for three to seven years. One patient is a professional swimmer and diver. He persisted in the sport and, of course, did not get any better. Another important factor to consider is, that of these 98 cases, over 90 per cent had had every type of treatment one could imagine, even short wave therapy, diathermy and zinc ionization. After all these types of treatments had been used without success, 91 per cent became cured by the use of the iodine powder. There is one fact, however, which I think should be impressed upon those who are gathered here, and that is this: I have noticed that some otologists and practitioners write a prescription for the powder and tell the patient to take it to the druggist and have it filled and then use it in the ear themselves. I recently had a serious case of threatened mastoiditis. The patient followed the physician's advice, but had his ear filled with the iodine powder, causing blocking of discharge. The result was almost a disastrous one. I think, therefore, the method should be restricted to the use of the otologist.

Recent Advances in the Study of the Hearing Sensation. Dr. John Guttman.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. EDMUND PRINCE FOWLER: This subject is intensely interesting to all of us. There are many pitfalls in the experimental work. The only way we will ever arrive at any conclusions is to keep at it. We must always remember that these potentials which we measure do not tell us what the animal is hearing. You can hook up electrical apparatus and get an electrical potential from an animal's big toe or any other place. I have tried to get these electrical effects from the round window of the human being and I thought I had succeeded several times. But in the end we found it was from some movement or reaction in the apparatus that the effect was obtained. I understand that someone abroad has recently obtained electrical effects from the round window but until further confirmation we must take this with a grain of salt.

The only way we will ever definitely find out how the cochlea works and how the nerve works is to persist in our experiments. Of course, even if we find that what Dr. Guttman has said is true, we have not got at the fundamentals as to what produces this change of electrical potential. About 15 years ago I was working with the Piezzo-electrical effects of Rochelle salt crystals and the thought came to me that possibly the end organs in the semicircular canals acted by this Piezzo-electrical effect. Of course, in the cupolae of the ampullae of the canals you have salts but it is difficult to find them in Corti's organ.